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Newer Oral Drugs for Diabetes

Although discovery of insulin has been one of the major blessings of medical progress, until recently there has been no effective method of controlling diabetes by an orally administered drug. With the discovery in Germany that carbutamide had a definite and prolonged hypoglycemic action, the door was opened to research on other compounds which might be more effective and less toxic. Strangely, carbutamide had caused few serious reactions in Europe, but when it was introduced into America, there was a sufficiently high incidence of severe toxic reaction, and even some deaths, to cause withdrawal of the drug from clinical use.

A new sulfonyl urea, tolbutamide (Orinase—Upjohn), was found to be much safer and possibly more effective. Widespread clinical use of the drug has not presented a notable problem of toxicity and has been a boon to many diabetics. Its usefulness, however, is restricted largely to those whose diabetes came on after maturity, whose insulin requirements are modest, and who are well controlled with insulin. The "brittle" diabetic whose hypo-hyper-glycemic swings persist and who may have periods of acetonuria in spite of careful efforts at dietary and insulin management usually is not helped by tolbutamide. It has been a useful tool, however, particularly in the older, stable, fairly mild diabetic whose eyesight, insight, tremor, or other disabling factors made insulin therapy a burden to himself or made him dependent upon a relative for his daily injection.

Even more recently, a different sulfonyl urea called chlorpropamide (Diabinese—Pfizer) has been under intensive study. It is not deactivated in serum and is slowly excreted. It reaches a peak effectiveness after 3 or 4 hours as

does tolbutamide, but its effective lowering of the blood sugar is considerably longer. Thus, a priming dose is not needed, no more than 0.5 Gm. daily should be given as an initial dose, and .025 Gm. every day or, in older mild diabetics, even every other day is often sufficient for control.

Like tolbutamide, the principal indication is adult diabetes mellitus which is uncomplicated, nonketotic, stable, and mild to moderately severe. However, chlorpropamide is sometimes effective in stabilizing "brittle" adult diabetes and permitting smoother control and less insulin.

A promising development not yet on the commercial market is a departure from the sulfonyl urea group. It is N^1-B-phenethylformanidinyliminourea (DBI—U.S. Vitamin Corporation). This preparation seems to have some effect on juvenile diabetes though work to date indicates that complete replacement of insulin or other hypoglycemic agents is possible only in the adult, stable diabetic. Unlike other compounds, DBI has a definite effect on juvenile diabetics of the "brittle" type. It sometimes will permit much better stabilization of these hard-to-control diabetics.

The dosage is smaller than with other drugs, but frequency of dosage is greater. One hundred to 150 mg. per day in divided doses with meals is the usually effective dosage range. Larger doses will produce frequent gastrointestinal symptoms. As little as 25 mg. daily may be effective in milder cases. There have been no toxic effects noted in the blood, liver, kidney, spleen, nor any other organ.

Thus, as is so often true in modern medicine, opening a door to research and finding one effective drug for a condition leads rapidly to new and better ones. The discoveries so far made in oral drugs are exciting and are very helpful to a sizable segment of diabetic patients. Tomorrow promises greater advances.

—MERTON M. MINTER, M.D., San Antonio.

Orientation Program

A short time ago the officers and members of the House of Delegates of the Texas Medical Association developed a program whereby provisional members, as a part of their education in the organization and tenets of American medicine, were introduced to the workings of the state organization through its central office and medical library and through seeing some of the elected leaders of the organization in action.

The reason for the program was twofold. It had been clear that some doctors enter the profession without recognizing the importance of relationships between physicians and their patients, physicians and their professional colleagues, and physicians and their medical organizations. Furthermore, prior to this period, interest of members of the Texas Medical As-

sociation had dropped to an all time low. It was believed that the best method of emphasizing the need for ethical, courteous, and mutually productive relationships and of helping to rekindle the fire of enthusiasm for organized medicine was through a program of education. A few county societies had a good educational and orientation set-up for their new members; however, at the state level there was no such program.

Many new members and a goodly portion of old members had begun to feel that they had little representation at the state level. It was felt that the central office at Austin was not a tool to be used by all members, but instigated and promulgated programs and ideas that were developed by a few. How better can such ideas be dispelled than to acquaint members with the physical properties of the central office and allow them to meet the staff of this office as well as physician officers and committee chairmen? Basically, then, the orientation program was planned to be held in Austin. With physical properties which equal those of any state organization in the nation there was adequate space to hold such programs. With the excellent personnel, whose sole function is to carry out the work of the Association, such a program was certain to succeed.

A wide range of subject matter was available for presentation; however, in the interest of time, it has been necessary to use only those topics which have a basic and direct bearing on a doctor's membership in medical organizations. At each of these meetings at least one outstanding speaker of national and often international repute has been featured. These presentations alone have been well worth the time and effort that our members have spent in attending these programs. In addition to these special features, much time and study has been devoted to the preparation and presentation of other subjects. This gives a picture of the internal affairs of the Texas Medical Association which no editorial or article could ever convey.

It should be pointed out that the orientation program, while designed for the new member, is of great interest to all who attend. Many doctors who have been present for the program have expressed the opinion that all members of the Texas Medical Association should attend such a program. Any person realizes that the more he contributes to an organization, the more benefit he derives from it.

Some criticisms have been leveled at this program; but in a recent survey conducted by the Board of Councilors, it was found that more than 70 per cent of those who had attended the program felt it was of great benefit to them. Conceivably, in the years to come such a program will be of such value to the membership that each member would profit from periodic attendance.

—CHARLES E. OSWALT, JR., Fort Stockton.

Specialists Also Serve

In a recent issue of the *Journal of the American Medical Association* there was an interesting article by Dr. Iago Goldston on "The Birth and Death of Specialties."* Basically, the text of the article is that with the advance of medical science, diseases will be controlled to the extent that by the year 2,000 there will be little specialism left. The article seems subtly to oppose specialism on the basis that few specialists will be needed in the future and that the specialists at the present time have little

*Goldston, I.: Birth and Death of Specialties, J.A.M.A. 167:2056-2061 (Aug. 23) 1958.

MERRY CHRISTMAS

to do with advancement of medical knowledge and the prevention of disease—only with the treatment of disease.

It is agreed that medicine is making tremendous strides, but it seems a bit Utopian to assume that by the "second millenium" most of the "ails that flesh is heir to" would be so well conquered that the medical practitioner can easily diagnose all conditions and readily prescribe the necessary pill to counteract them. Surely there will be enough cases difficult of diagnosis and stubborn of treatment to warrant specialists in the various fields, who could devote their entire time and attention to such individual problems.

The average present day specialist should not, and usually does not, consider himself a competitor of the general practitioner, but rather as someone to aid the general practitioner in the diagnosis and treatment of disease. He is servant both to the patient and the referring physician, and he will be derelict in his obligation to both if he allows his observations and deductions to be too closely confined to one field. The successful specialist (as well as the successful general practitioner) is the one who remembers that each system is but a part of the whole, and that malfunction in one system may reveal itself as symptoms apparently arising in another area.

Dr. Goldston says, "A specialist is one who does not practice comprehensive medicine. He only exercises his skills in some subdivision or in some partial function within the larger framework of medicine." If I may be allowed to correct this statement, it should read: "A *good* specialist is one who *practices* comprehensive medicine, but *exercises* his skills only in some subdivision . . . within the larger framework of medicine."

Exception must be made also to the idea that the specialist's only interest is in *treatment* of disease. All will agree that the future of medicine lies in research, experimentation, and investigation of the physiological aspects of medicine. There are large numbers of specialists engaged in research in physiology and pathology, as well as in treatment. It is more than a coincidence that the increase of specialization is concomitant with the advancement of medicine.

There can be no doubt that specialism is a pendulum that has swung far out and is now swinging back a bit. This is as it should be. For a while few medical students thought of doing a general practice following their graduation—all were interested only in specialties. Now a higher percentage of men are going into general practice because the honor and stature of the title "G.P." is being given the value it deserves. The past and future of medicine depends on the general practitioner as being the keystone, but there must also be specialists to work hand in hand with the practitioner to the common goal of bringing better care to all people.

—LOUIS J. LEVY, M.D.,† Fort Worth.

†*President, Texas Orthopedic Association.*

Open to All Members—

Texas Medical Association
Conference of County Society Officials
Symposium on Legislation, and Orientation Program
January 24, 1959—Austin

Executive Council—January 25, 1959

—See Organization Section for Details

President's Page

THE WOMAN'S AUXILIARY

DR. SMITH

The Woman's Auxiliary to the Texas Medical Association, which now has more than 5,500 members, has been of inestimable help to the Association throughout the 40 years of its existence. The intangible results of our having the faith and confidence, the ever present thoughts and loyalty, and at times guidance of our ladies is irreplaceable. They have never failed us.

The constructive projects of the Auxiliary are many, paralleling the program and objectives of the medical association. Of special interest to physicians, perhaps, are the philanthropic funds of the Auxiliary, which provide support of the Association's Memorial Library (which the women use themselves, especially the motion picture film division), loan funds for medical students, and gifts to families of deceased physicians who are in need. Another big financial objective is the American Medical Education Foundation, for which the Texas Auxiliary won recognition last year for the largest collection of any state auxiliary. At latest report more than 40,000 Christmas cards had been sold for the AMEF project this year. The card bears an original scene of the Big Bend region sketched by Mrs. D. J. Sibley of Fort Stockton, a member of the Auxiliary.

Recruitment is one of the greatest success stories of the Auxiliary. Future Nurses Clubs are sponsored in high schools over Texas by the Auxiliary, nursing organizations, and school sponsors—teachers or nurses. To date there are about 230 clubs with a student membership exceeding 5,000. In these clubs the young people learn not only about nursing, but also about other types of health careers.

Closely related to recruitment are the science fairs, which encourage youngsters in high school to exhibit their inventions or results of study along scientific lines whether medical or not. County auxiliaries in many areas sponsor local science fairs and provide funds to send representatives to state and national fairs. Two top winners in the national competition last spring came from Austin.

Safety and civil defense are other emphases of the Woman's Auxiliary. Safety in the home, on the highway, in recreation; safety in

the use of baby sitters; safety in relation to possible poisoning of children—all these are being taught through the Auxiliary. Members of medical auxiliaries are learning techniques of civil defense and participating in civil defense organizations. They are encouraging the showing of the film, "The H-Bomb," available on loan from the Texas Medical Association Library.

Today's Health, the only magazine endorsed by the medical profession for laymen, published by the American Medical Association, is distributed in Texas partly through the Woman's Auxiliary, which encourages subscriptions for its own members, doctors' offices, and other places where magazines are read and re-read. In further efforts to help the public be health conscious, the Auxiliary has distributed 300,000 personal health cards and 100,000 health record booklets.

Our legislative program always finds members of the Woman's Auxiliary eager to learn about bills of health import and ready to spread the word about action that will be of benefit. This aspect of Auxiliary activity will be brought into play again the next few months when both the Texas Legislature and the national Congress will be in session and measures of special interest to the medical profession will be under consideration.

News of Auxiliary projects is distributed to all members of the Auxiliary in a *News Letter* published bimonthly by the Auxiliary, and information of interest to doctors is carried regularly in the *Journal.* Too, the Auxiliary President, Mrs. John D. Gleckler of Denison, has been visiting all parts of Texas not only to learn firsthand what the county auxiliaries are doing but to tell doctors and their wives what the Auxiliary is achieving and hopes to achieve.

In 700 B. C. one of the prophets of Nebuchadnezzar said: "Cursed be he that doeth the work of the Lord negligently." One can never say that our ladies do their work negligently. Our unstinted support of them will be advantageous to us all.

Now, before closing this month's message, let me extend to each member of the Texas Medical Association and its Woman's Auxiliary best wishes for the holiday season and for the coming year.

Howard O. Smith M. D.

ORIGINAL ARTICLES

The Problem Of Recurrent Incisional Hernia

AMOS R. KOONTZ, M.D.

Baltimore, Maryland

FOLLOWING the operation for cure, incisional hernias, like inguinal hernias, recur entirely too frequently. There would be no problem of the recurrent incisional hernia if there were no incisional hernias in the first place. Most of them could be prevented.

PREVENTION OF INCISIONAL HERNIAS

In the prevention of incisional hernias, the type of abdominal incision is considered highly important by a great many people. I believe, however, that the method of closure is more important than the type of incision. Vertical incisions are much more widely used than transverse incisions, and probably will continue to be, because of the wider range of exploration possible through them. The number of incisional hernias occurring through vertical incisions is much greater than those occurring through transverse incisions. One reason for this is certainly the fact that the vertical incision is much more widely used. A recent survey of a series of cases showed that the percentage of incisional hernias occurring in transverse and vertical incisions was approximately the same. McVay,[12] however, has presented a strong case for the transverse incision. He showed that the majority of aponeurotic fibers in the transversus abdominus and internal oblique muscles run in the transverse direction and that these fibers naturally tend to close a transverse abdominal incision rather than open it, which is not the case in vertical incisions.

No matter what type of abdominal incision is chosen, it is highly important to close it properly. Catgut should never be used, except in the presence of infection, because it has been demonstrated many times that catgut sutures are not reliable beyond a few days[11] and it is well known that it takes 10 to 14 days to get good healing in any wound.[2] Therefore, to use catgut sutures in closing an abdominal incision is to invite incisional hernia by the absorption of the sutures before healing takes place. The wound should be closed carefully in layers. I prefer a continuous silk suture in the peritoneum, because it eliminates small gaps in the peritoneal closure through which small tags of omentum may protrude if interrupted sutures are used. The abdominal fascia should be closed by a true fascia-to-fascia closure. This cannot be done if areolar or adipose tissue is left on the fascial edges when they are sutured together. These extraneous tissues can be eliminated if, in making the abdominal incision, the fascia is cleared of adipose and areolar tissue in an area at least ½ inch wide before incising the fascia. This practice makes it easy to close the fascia with interrupted sutures without the interposition of tissues which will prevent good healing and thus contribute to incisional hernia.[4]

There is considerable difference of opinion as to the advantage of stay sutures. I no longer use the old type of through-and-through stay sutures including all layers of the abdominal wall. When the fascia shows evidence of weakness, I sometimes use buried far-and-near stay sutures of the type shown in figure 1. These distribute the tension on weak fascia and tend to prevent the separation of the fascial edges.

CAUSES AND CURE

Incisional hernias sometimes occur even in the best of hands. The causes when the abdominal wound

Dr. Amos R. Koontz, assistant professor emeritus of surgery at Johns Hopkins University School of Medicine, presented this paper before the Texas Traumatic Society on April 21, 1958, in Houston.

Most incisional hernias can be prevented by proper closure. Once they occur, operation for cure should be performed early. Relaxing incisions; far-and-near tension sutures; flaps of reinforced peritoneum; reinforcing agents such as tantalum gauze, cutis grafts, or fascia transplants; and preoperative pneumoperitoneum are valuable in prevention and cure.

has been properly closed are generally postoperative distention, coughing, or vomiting. However, most incisional hernias are due to sloppy closures of abdominal wounds. Once they have occurred, they should be operated upon as soon as possible. The following paragraphs deal with some of the causes of failure and some of the aids in the operation for cure.

Late Operation.—Every incisional hernia should be operated upon as soon as possible after it is discovered. It generally is easy to cure in the early stages and often difficult to cure after it has been present a long time. A great many incisional hernias enlarge fast after they first appear. Abdominal organs protruding through the sac push the muscle and fascia in all directions until a large defect has developed. Incisional hernias, especially those in the epigastric region, are often among the most difficult of all hernias to cure. Hence, it is important to attack them early when both the hernia and the surgical problem are small. The surgical problem increases in direct proportion to the size of the hernia.

Improper Closure.—I operate on a great many incisional hernias which have been operated on several times before. As a rule, they have been operated upon once or twice by the surgeon who made the first abdominal incision. Often an attempt is made to counterbalance the effect of a sloppy closure by the use of wire as a suture material. Then when the hernia recurs, the surgeon says to the patient, "I don't see how that could have happened, I used wire in operating on you the last time." There is no magic in wire or in any other suture material. The magic is in the hands and the brain of the surgeon. Wire improperly used is nothing like as good a suture material as silk, because it tends to cut through much more readily than silk. Its only advantage is that it does not cause as much trouble in the presence of infection as silk does. However, I have seen persistent sinus tracts from heavy wire improperly used in abdominal closure.

Relaxing Incisions.—In the case of large defects, the edges of which are difficult to approximate, relaxing incisions should be made in the fascia over muscles adjacent to the defect whenever possible. In incisional hernias through rectus incisions, as a rule it is possible to make relaxation incisions in the sheath of the rectus on each side, which greatly facilitate closure. If the muscle is good under the incisions, one need not fear the occurrence of a hernia through the muscle. It has been demonstrated experimentally that the sheath of the rectus regenerates.[5] However, relaxation incisions should not be made through fascia under which there is no muscle. If this is done, a new hernia occurs at the point of the relaxation incision.

Far-and-Near Stay Sutures.—In the presence of large defects, even after relaxation incisions have been made often the fascial defects have to be approximated with some tension. Ordinary interrupted sutures placed under tension tend to pull out. My practice is to use far-and-near tension sutures of number 4 braided white silk as shown in figure 1. These sutures distribute the tension and effect a good and easy approximation of the fascial edges. The fascial edges should not be dissected into layers, but the entire full thickness of the abdominal wall approximated. Dissecting structures into layers weakens the abdominal wall and the repair. After the far-and-near sutures are placed, at times the muscle under the relaxing incisions separates as the result of tension. If the separation is in the upper abdomen where there is a good posterior sheath of the rectus, little damage is done. However, in such instances I generally place either a piece of tantalum or a cutis graft under the muscle at the point of separation. This has proved to be a satisfactory procedure.

Flaps of Reinforced Peritoneum.—In most incisional hernias the sac is not composed simply of

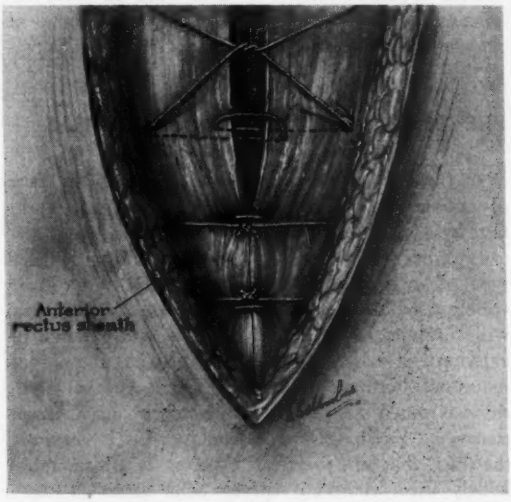

Fig. 1. Far-and-near traction suture nicely approximates the fascial edges in cases in which there is some tension. It also is useful in cases in which there is no tension, but in which the fascia is poor and in which sutures in the fascial edges themselves are likely to pull out. The suture distributes the tension over a wide area of the fascial plane instead of leaving it simply at the fascial edges. Heavy silk is used because it does not tend to cut through as silk of a smaller caliber does. The notion that no suture should be used any stronger than the structure sutured is fallacious in this respect. No matter how strong a small suture is, if it cuts through, it defeats its purpose. Between the tension sutures, interrupted sutures of a smaller caliber silk are used. The traction suture is not original with me but, so far as I know, I was the first to use it as a buried tension suture. [After Koontz, A. R.: Failure with Tantalum Gauze in Ventral Hernia Repair, A.M.A. Arch. Surg. 70:125 (Jan.) 1955.]

peritoneum, but of peritoneum reinforced by fibrous tissue and scar tissue. In those cases in which the edges of the defect cannot be approximated (rare if proper relaxing incisions are used), flaps may be fashioned out of the reinforced peritoneum and overlapped to close the defect. This may be done by simple overlapping or by using Johns' ingenious method of interlacing tongues of tissue prepared from the reinforced flaps.[8]

Reinforcing Agents.—After the defect has been closed as well as possible with the materials at hand, it often is thought that the closure should be reinforced, either because the closure has been made under tension or because the patient's tissues are so poor that it is thought they will not withstand the force of intra-abdominal pressure. Various reinforcing agents have been used such as tantalum gauze, cutis grafts, transplanted fascia, and, lately, lyophilized dura mater.[14] I have used the first three but not the last mentioned. I now prefer tantalum as a rule, because it is ready and does not have to be prepared, thus prolonging the operation. Besides, cutis grafts and fascial transplants cause trouble in the presence of infection while tantalum does not.[7]

If flaps of reinforced peritoneum have been fashioned to close the defect without actually approximating its fascial edges, it is important to reinforce the area, which I do with tantalum. Also, if the structures are poor, reinforcement is indicated. Often in fat people the fascia is so poor that fat literally can be seen oozing through the interstices of the fascia. Closure of a defect with such weak structures will not cause a permanent cure. Reinforcement should be effected. In several cases in which I used a small piece of tantalum, a new hernia occurred at the edge of the tantalum because the tantalum had not been carried far enough to cover the weak structures adjacent to the hernial area. Reinforcement also should be used in those cases in which relaxation incisions have been made and the defect closed under so much tension that the muscle under the relaxation incisions separates a good deal. This is especially important in those patients who have a weak musculature and weak structures in general.

Smith[13] has properly pointed out the disadvantages of the use of tantalum in large incisional epigastric hernias. As stated before, these hernias are among the hardest to cure. If the fascial edges cannot be approximated and one depends simply on flaps of reinforced peritoneum to close the defect, tantalum works effectively as a supporting material for a time. The eventual fragmentation may cause a recurrence in this location, however, when it will not do so if similarly used in the lower part of the abdomen. The stresses are certainly greater in the epigastric region. Nevertheless, if the fascial edges can be approximated although under tension, the reinforcement with tantalum is a great boon. Fragmentation after firm fascial healing has taken place is no deterrent to the cure. By the time fragmentation takes place, the interstices of the tantalum have been so thoroughly interwoven with firm fibrous tissue that the fragmentation makes no difference. The object of the tantalum is not to cure the hernia per se, but simply to act as a framework for the ingrowth of fibroblasts. It has been shown that tantalum actually stimulates the growth of fibroblasts, and does not lie entirely inert in the tissues as does stainless steel.[10]

In the epigastric region when the fascial edges cannot be completely approximated and one has to resort to flaps of reinforced peritoneum, I think it is better to use cutis grafts or fascia as a reinforcing agent for the reasons mentioned in the last paragraph. I personally prefer cutis grafts. Whole thickness skin grafts are fine if small grafts are used, but when large grafts are necessary, they are often troublesome because of the slowness of the absorption of the epithelial elements and the accumulation of fluid.

Pneumoperitoneum.—A great many incisional hernias are so huge that most of the abdominal contents are in the hernial sac and have forfeited the right of domicile. In such cases preoperative pneumoperitoneum makes an impossible operation possible. This valuable method, first described by Goñi Moreno of Buenos Aires,[1] may be simply done. I use a large syringe with a 2-way stopcock and 19-gauge lumbar puncture needle. Air is injected into the peritoneum until the patient complains of respiratory distress or other symptoms, such as pain in the shoulder. The initial amount injected varies from 500 cc. to 3,000 cc. Additional injections are given every 3 to 4 days until the peritoneal cavity has been enlarged enough to receive the expatriated abdominal contents. I have injected as much as 18 liters over a period of 2 or 3 weeks. During the course of injections an abdominal binder should be worn in order to prevent the air from becoming captive in the hernial sac and not enlarging the peritoneal cavity. Once the peritoneal cavity has been enlarged sufficiently, it is amazing how simple is an operation which at first looked impossible. When the incision is made into the peritoneum, the air escapes with a "poof." The abdominal walls are found to be flaccid, the abdominal contents are easily reduced, and the repair is made much easier.[8, 9]

SUMMARY

Most incisional hernias can be prevented by proper closure at the time of the primary operation. Once they do occur, the operative cure should be performed

early. The longer the operation is postponed, the larger the hernia and the more difficult the cure. There is no magic in wire, which some surgeons use to cover up a faulty closure. Relaxing incisions should be used freely in order to make possible the approximation of the edges of the defect. Far-and-near tension sutures should be used in approximating the edges in order to distribute the tension over wide areas of the fascia instead of having it simply at the edges, as is the case when interrupted sutures alone are used. In those cases in which the edges of the defect cannot be approximated, flaps of reinforced peritoneum may be fashioned to close the defect. When the closure is weak for any reason, some reinforcing agent such as tantalum gauze, cutis grafts, or fascia transplants should be used. Preoperative pneumoperitoneum is an invaluable aid in those hernias which have forfeited the right of domicile.

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◗ Dr. Koontz, Department of Surgery, Johns Hopkins Hospital, Baltimore.

35,000 MD's Studied in 1958

More than 35,000 physicians last year took graduate medical training in 1,400 American hospitals, according to the annual report prepared by the American Medical Association's Council on Medical Education and Hospitals. There were 10,198 graduates serving internships in 1957-1958, an increase of 305 over 1956-1957, while 24,976 were serving residencies, an increase of 1,964 over the preceding year.

Treatment of Hemangioma of Infants and Young Children

ROBERT J. REEVES, M.D.

Durham, North Carolina

HEMANGIOMA OF THE SKIN or mucous membrane is a lesion familiar to every physician. It is a benign tumor or new growth of tissue in which there is actual new formation of vessels or there is a localized hyperplasia of the cutaneous tissue or both. The lesions may involve the dermis, the subcutaneous tissue, or both. Sharp distinction must be made between vascular enlargements which are varicosities or aneurysmal dilatations of preexisting blood vessels, self-limited fibroblastic and endothelial hyperplasias, and true hemangiomas which are embryonic sequestrations of unipotent angioblastic cells. The origin of hemangiomas therefore can be traced to the failure of these cell groups to establish contact with the developing network of blood vessels. Growth is thought to be accomplished by budding of vascular channels and proliferation of endothelial cells.

The classification of nevi may be made on an anatomic basis. That made by Pack and Miller, however, is probably the most complete. Their listing appears in table 1.

My classification depends on whether the lesion is relatively sensitive or resistant to irradiation and whether this treatment is definitely indicated.

Dr. Robert J. Reeves, professor of radiology at Duke University School of Medicine, presented this paper for the Section on Radiology of the Texas Medical Association at the 1958 annual session in Houston, April 21.

This study based on 400 cases of hemangioma of the skin, some of which have been followed as long as 20 years, suggests that treatment by radium and roentgen irradiation alone or in combination may give good results. A classification of hemangiomas according to their sensitivity or resistance to irradiation is given.

The group which is relatively sensitive to irradiation includes the following:

1. Cavernous hemangiomas. These are composed of large blood spaces mostly veins and venules with minimal arterial element. They may be in the cutis or subcutis, may be irregular or circumscribed lesions, and may vary in size from a pinhead to very large lesions. They may have bluish or reddish color, and are usually compressible. The skin covering the lesion may be normal and movable or atrophic and adherent to it.

2. Strawberry hemangiomas. These are capillary lesions, usually elevated, irregular in shape, of relatively rapid growth, and light red in color and have a sensitive-to-irradiation epithelial lining.

The cavernous and strawberry types are the most sensitive to irradiation.

3. Mixed cavernous and strawberry hemangiomas. These consist of both elements described previously.

4. Bright red capillary nevi. These are nonelevated lesions from which the color can easily be pressed out and are thought to be sensitive to irradiation in contrast to the dark red type (port wine) which are resistant to irradiation.

5. Spider nevi. These are also capillary lesions which vary in size. They may increase in size at any time by proliferation of the endothelial layer forming radiating vessels from the elevated central vessel and are considered by some observers to be relatively sensitive to irradiation.

The group which is relatively insensitive to irradiation includes:

1. Port wine stain lesions. These are of the capillary type and are flat, pink or purplish in color. They may be diffuse and superficial in the dermis and consist of venules and capillaries which grow pari passu with the child. These often persist and become thicker. They are lined by an adult type endothelium which is resistant to irradiation.

2. Hemangioma simplex. These are blotchy areas often found on the skin of the neck which may disappear spontaneously, and in these, treatment is not usually indicated.

3. Sclerosing hemangiomas. These are hard brown or bluish lesions just under the skin, which are seldom larger than 1 cm. in diameter and in which connective tissue proliferation predominates and the lumen of the vessel is often invisible.

Most of the cases I have treated in the last 20 years were of the cavernous and strawberry types. Only a few capillary or spider nevi were treated.

TABLE 1.—*Classification of Nevi.*

Tumors of Blood Vessels
Capillary hemangioma:
Simple type.
Port wine stain (nevus vinosus).
Nevus araneus. Nevus flammeus. DeMorgan spots.
Sclerosing hemangioma.
Infectious hemangioma:
Pyogenic granuloma.
Granulation cell sarcoma.
Cavernous hemangioma:
Superficial type.
Visceral type.
Hypertrophic hemangioma:
Simple type.
With metaplastic elements.
Cirsoid or racemose hemangioma:
Arteriovenous fistula.
Systemic hemangiomatosis or hemangioma unius lateralis:
Congenital.
Acquired.
Hereditary hemorrhagic telangiectasis (Rendu-Osler-Weber's disease).
Congenital neurocutaneous syndromes associated with angiomatosis:
Von Recklinghausen's neurofibromatosis and angiomas of the skin.
Bourneville's syndrome with tuberous sclerosis.
Pringle's disease and regional angiomas.
Sturge-Weber's disease. Encephalofacial angiomatosis.
Lindau-von Hippel's disease. Hemangiomatosis of retina and cerebellum.
Hemangiopericytoma:
Benign glomus tumor (Masson).
Malignant type (Strout's tumor).
Special regional hemangiomas:
Orbit and eye.
Brain.
Tongue.
Gastrointestinal tract.
Liver.
Skeletal muscle.
Bone.
Malignant tumors of blood vessels:
Angiosarcoma.
Endothelioma.
Hemangiopericytoma.
Kaposi's idiopathic sarcoma.

Compiled from data in Pack, S. T., and Miller, T. R.: Hemangiomas, Angiology 1:405-426 (Oct.) 1958.

CURRENT STUDY

This study includes 400 cases of hemangioma from a group of 939 patients who were treated by radium or roentgen irradiation or both and in whom a long period of follow-up was possible either from my records or by communicating with the families. Table 2 shows the race and sex distribution of patients. Table 3 shows the age distribution.

TABLE 2.—*Race and Sex Distribution of 400 Patients with Hemangiomas.*

Females		Males		
White	Negro	Indian	White	Negro
282	17	1	90	10
70.5%	4.25%	0.25%	22.5%	2.5%

TABLE 3.—*Age Distribution of 400 Patients with Hemangiomas.*

Newborn-1 yr.	1½-2 yr.	2½-5 yr.
322	55	23
80.5%	13.75%	5.75%

THERAPEUTIC CONSIDERATIONS

For many years there have been many physicians who do not recommend treatment of capillary or cavernous hemangiomas, largely because they have been taught that most of the hemangiomas will disappear spontaneously. There is no doubt that some of these lesions do heal spontaneously, but it is not possible to predict with assurance which lesions will remain quiescent or disappear spontaneously. I have seen many hemangiomas in adults, many on the faces of young women.

The methods of therapy for the various types of cutaneous hemangioma have been given as (1) surgical excision; (2) roentgen therapy; (3) radium therapy; (4) carbon dioxide snow; (5) injection of sclerosing solutions; (6) tatooing and/or cosmetic disguise.

For many years I have used radium molds and plaques, filtered and unfiltered, for one-third of an erythema dose. It is a commendable form of therapy and many excellent results were obtained. For the past 12 years, however, I have used 100 Kv. roentgen ray almost exclusively. Many of the older radiologists overtreated these cases. Kaplan advocated 100 Kv., 0.5 mm. aluminum filter, with a dose of 200 to 250 r once weekly for 3 to 5 weeks. Some of these patients showed later evidence of overtreatment.

The factors I have used are 100 Kv., 10 cm. distance, 1 mm. aluminum filter. A dose of 200 to 250 r is given and repeated in 2 months and again at 6 months if there is no regression.

Hemangiomas are more sensitive to irradiation during infancy and early childhood than in later years, but so are the normal tissues, especially bones, where they are in close proximity. I am especially careful over the joints or near the tooth buds. The principle of therapy is to obliterate the tumor by sclerosing fibrosis of the tumor. The predictable success of radiation therapy cannot always be made. The immediately good after result is sometimes spoiled by late atrophy of the skin, subcutaneous tissue, or bone. It may not be evident until 10 or 15 years later. Radiation telangectasia developing in the facial skin of children or young adults treated in infancy may require surgical excision and plastic repair. Hemangiomas near the breast of female infants or external genitals of boys should be treated surgically. If hemangiomas are near the eye, a preliminary ophthalmological examination is wise as a precaution in order to be certain about defects in the eye related to the tumor. Roentgen-ray therapy about the eye is preferable to radium because of the ease of shielding and lesser danger of cataract or glaucoma, secondary to radiation iridocyclitis.

The deeply situated angiomatous masses can be treated interstitially by intratumoral injection of 1 millicurie radon seeds. The injection is made through normal skin margin to prevent excessive bleeding.

COURSE AFTER TREATMENT

In this series follow-ups from 1 to 20 years were obtained in all but 37 cases.

Results of treatment as shown in tables 4 and 5 are classified as follows:

1. Satisfactory results: When lesions disappeared completely, leaving no scar or a very small one, or lesions showed a marked bleaching, flattening, or decrease in size and were considered to need no more treatment.

2. Fair results: When lesions showed partial changes in color, size, or thickness and more treatment was considered indicated to achieve better results.

3. Poor results: When lesions were resistant and progressing in growth, and surgery was indicated as a final resort.

TABLE 4.—*Follow-Up Results of Treatment of 400 Cases of Hemangioma.*

Period of Follow-Up	No. of Patients	Satisfactory	Fair	Poor
9 mo.-1 yr.	128	75(60%)	46(35%)	7(5%)
1½-5 yr.	202	151(75%)	39(19%)	12(6%)
5½-10 yr.	53	43(81%)	8(15%)	2(4%)
12-20 yr.	17	15(88%)	1 (6%)	1(6%)
Total Results		284(71%)	94(23.5%)	22(5.5%)

Most lesions require more than 1 year to give a satisfactory result. The longer the period of follow-up, the higher is the percentage of satisfactory results.

Table 5 shows the results of treatment according to the type of treatment used. There was complete failure in 3 to 5 per cent in the various groups. A high percentage of failure is shown in the roentgen irradiation alone. Of 7 cases of port wine lesions only 1 showed complete clearing, but latent skin damage developed. In the adult group the results generally are poor and surgery is always advised.

TABLE 5.—*Follow-Up Results According to Type of Treatment.*

Type of Treatment	No. of Patients	Satisfactory	Fair	Poor
Radium	198	162(81.9%)	33(16.6%)	3(1.5%)
Roentgen irradiation	124	110(88.7%)	14(11.3%)	0
Both	78	45(57.7%)	28(35.9%)	5(6.4%)
Total*	400	317(79.3%)	75(18.8%)	8(2%)

*Percentage more than 100 because of rounding off fractions.

A higher percentage of satisfactory results were obtained by radium therapy alone; however, a larger group was treated with this method, and there is a longer period of follow-up on most of them. This kind of treatment was more frequently used in the earlier years of my experience, and the difference between radium and roentgen irradiation is not considered significant.

Of the 400 patients, 18 have developed scarring in the period of 2 to 8 years after treatment. Seven of these were treated with radium, 4 with roentgen therapy, and 7 with a combination of radium and roentgen ray. One of the scars caused marked puckering of the lip. Four patients developed telangiectasis; 1 patient had shortening of the leg and atrophy of the muscles of the treated leg. In 4 patients new hemangiomas developed after treatment, either at the same site or in new areas. In 10 patients, surgery was done for either cosmetic reasons or failure of irradiation. In 2 patients, intracranial aneurysm coincided with the hemangioma on the face (Sturge-Weber syndrome).

SUMMARY

A group of 400 cases of hemangioma of the skin were studied and follow-up results are given. The majority were of the cavernous and strawberry types. The classifications given are according to the relative sensitivity to irradiation. Various groups are separated depending on the type of treatment used.

The sites of predilection are the head and face. Complications are not frequent if treatment is given carefully.

Both radium and roentgen irradiation give good results, and a combination of both may give no better results than either alone. Interstitial radon seeds may be used with good results in large and deep lesions, but none were treated as such in this group. I consider either radium or roentgen irradiation as acceptable modes of treatment and believe them to be superior to other methods of therapy.

This series of cavernous and strawberry type lesions was not broken down as to results. It is true the small strawberry type frequently will disappear spontaneously and a number of cavernous ones may do likewise.

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Hypercortisonism In Patients with Rheumatoid Arthritis

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THE SYNDROME of "hypercortisonism" as seen in patients with rheumatoid arthritis under long term adrenal steroid therapy was first described by Slocumb.[9, 10] In addition to the usual features of Cushing's syndrome which may be induced in any patient by the administration of adrenal steroids, an additional group of features was described. Since then other observers have reported recognizing this syndrome in patients with rheumatoid arthritis. It is the purpose of this paper to reemphasize the importance of these special features of hypercortisonism in general and to describe the vascular lesions occurring in a recent series in particular.

CLINICAL PICTURE

The clinical features of Cushing's syndrome which may be produced in patients receiving adrenal steroids are now familiar to most physicians. These features include fluid retention, moon face, abdominal striae, enlarged supraclavicular and cervicodorsal fat pads, hypertension, diabetes, osteoporosis, purpura, insomnia, hirsutism, excessive appetite, nervous

Dr. James W. Kemper, a member of the Kelsey and Leary Clinic staff, gave this paper at the Texas Rheumatism Association meeting December 6, 1957, in Houston.

Patients with rheumatoid arthritis treated with adrenal steroids may develop, in addition to features of Cushing's syndrome, a syndrome of cyclic mood changes, fatigability, and musculoskeletal aching. Also they may have "mesenchymal reactions" similar to lupus erythematosus, a flare of rheumatoid arthritis, or periarteritis nodosa. Treatment consists in steady, gradual reduction of dosage in small decrements.

tension, and other symptoms. It has been reported that from 66 to 75 per cent of all patients treated with adrenal steroids will develop one or more of these features.[8] In addition to these usual features, however, many patients with rheumatoid arthritis under long term adrenal steroid therapy have been seen to develop certain unusual or "special" features of hypercortisonism.[9, 10, 11] These special features comprise a syndrome which is characterized by musculoskeletal aching, fatigability, and cyclic mood changes. Because of the similarity of many of these features to those of rheumatoid arthritis itself, the patient and the physician both may mistakenly interpret these features as a flare-up of the arthritis. To emphasize this, Rotstein and Good have called this syndrome "steroid pseudorheumatism."[8] Careful questioning and evaluation, however, reveal definite distinguishing features between the special features of hypercortisonism and a true flare-up of the rheumatoid arthritis.

Whereas in a rheumatoid flare symptoms will be concentrated on the joints and some degree of synovitis will be present, the musculoskeletal aching of hypercortisonism is more diffuse and seems to involve muscles and bones with synovitis being minimal. The aching seems out of all proportion to the objective findings. While heat, physical therapy, and salicylates will help most rheumatoid flares, these measures have little effect on the aching of hypercortisonism.

The second feature is profound physical and mental fatigue. The cyclical nature of this fatigue and speed of its onset are striking. Patients may relate how they will suddenly become so utterly fatigued that they must stop whatever they are doing to lie down and rest. While fatigue generally is common in rheumatoid arthritis, it is not ordinarily of this nature.

The cyclic mood changes are characterized by periods of depression and inability to concentrate or remember, which will alternate with periods of euphoria, stimulation, and a general feeling of well-being. These fluctuations may occur within a few hours. Repeated observation and interviews may be necessary to obtain a complete picture of the patient's status, since an erroneous impression may be obtained from an isolated interview depending upon what mood the patient is in. When depressed the patient cannot remember details, is disinterested, and completely uncooperative. It may be extremely frustrating to try to obtain a detailed past history. The patient feels terrible and will admit to no beneficial effect of any past treatment. On the other hand, when euphoric, he may give an entirely different history, minimizing symptoms, manifesting a restlessness and stimulation, and will have nothing but praise for the merits of cortisone, the physician, and all concerned. In a rheumatoid flare mild depression may occur, but not the profound depression, lack of concentration, and impairment of memory just described, nor are the rapid fluctuations in mood ordinarily seen.

Many intelligent rheumatoid patients themselves will notice the difference between the special features of hypercortisonism and those ordinarily attributed to rheumatoid arthritis. Several have volunteered the observation that "this is something different from my arthritis" — "I never had this before."

In general the features described are most pronounced when 5 or 6 hours have elapsed since the last dose of steroid, or may be precipitated by missing a dose. Short periods of rest or a dose of steroid will cause the symptoms to abate temporarily only to return later. If the physician fails to recognize the chain of events and submits to the request of the patient for more steroid, repeated increments in dosage followed by ever recurring symptoms soon produce a difficult and dangerous situation.

In some patients with rheumatoid arthritis subjected to chronic hypercortisonism, more serious reactions have been seen to develop which seem to involve mesenchymal tissues and may present clinical findings similar to other disease entities. These have been termed "mesenchymal reactions."[10, 11] These reactions may simulate a flare of the rheumatoid arthritis, present clinical features of lupus erythematosus including a positive L.E. reaction, or appear as polyarteritis nodosa both clinically and by biopsy. Fever, increased erythrocyte sedimentation rate, hemorrhagic skin lesions, pleurisy, and peripheral neuritis, pericarditis, pulmonary consolidation, thrombophlebitis, and false-positive serologic reactions for syphilis also have been noted. So far, only patients with rheumatoid arthritis (with the possible exception of some with lupus erythematosus) have been seen to develop so-called "mesenchymal reactions."

Evidence of the apparent relationship of hypercortisonism to the production of "mesenchymal reactions" in patients with rheumatoid arthritis was provided by the study of Frerichs.[3] He reviewed the clinical records of all patients on whom an L.E. test was performed at the Mayo Clinic. The patients were divided into several groups according to the disease and the treatment received. In the group of patients with classic rheumatoid arthritis who had never been treated with cortisone, the incidence of positive L.E. reactions was 5 per cent. The same incidence was found in a similar group with rheumatoid arthritis who had been treated with cortisone, but showed no evidence of hypercortisonism. However, in the group with rheumatoid arthritis who had received cortisone and who had hypercortisonism, the incidence of positive L.E. reactions was increased to

14 per cent. In this group there was also a higher incidence of fever, albuminuria, transfusion reactions, leukopenia, serositis, and purpura.

Other patients have been seen to develop a clinical picture similar to polyarteritis nodosa with positive evidence of necrotizing arteritis on biopsy. To investigate the vascular lesions occurring in rheumatoid arthritis and their relation to adrenal steroid therapy, my associates and I reported earlier on a review of all instances of rheumatoid arthritis examined at necropsy at the Mayo Clinic through 1954.[5] Special emphasis was placed on the incidence and character of the vascular lesions and their relationship to the administration of cortisone. Strict criteria were used to insure the inclusion of only bona fide cases of classic rheumatoid arthritis. There were 52 patients in this group of whom 14 had received cortisone. Four of these 14 patients had lesions of a diffuse necrotizing arteritis indistinguishable from polyarteritis nodosa (fig. 1). None of the 38 patients not having been treated with cortisone had such lesions. Of the 4 with the necrotizing arteritis, definitely 2 and probably 3 were suffering from the syndrome of hypercortisonism described previously. It is important to emphasize that these vascular lesions were not manifested by an occasional arteriole with perivascular lymphocytic infiltration, or a little thickened collagen, but acute necrotizing inflammatory lesions with cellular exudation and fibrinoid necrosis involving almost every organ and tissue examined. In 3 of the 4 patients the vascular lesions were responsible for the patient's demise. In 3 of the 4 patients peripheral neuritis in the form of a mononeuritis multiplex was a prominent finding, and in

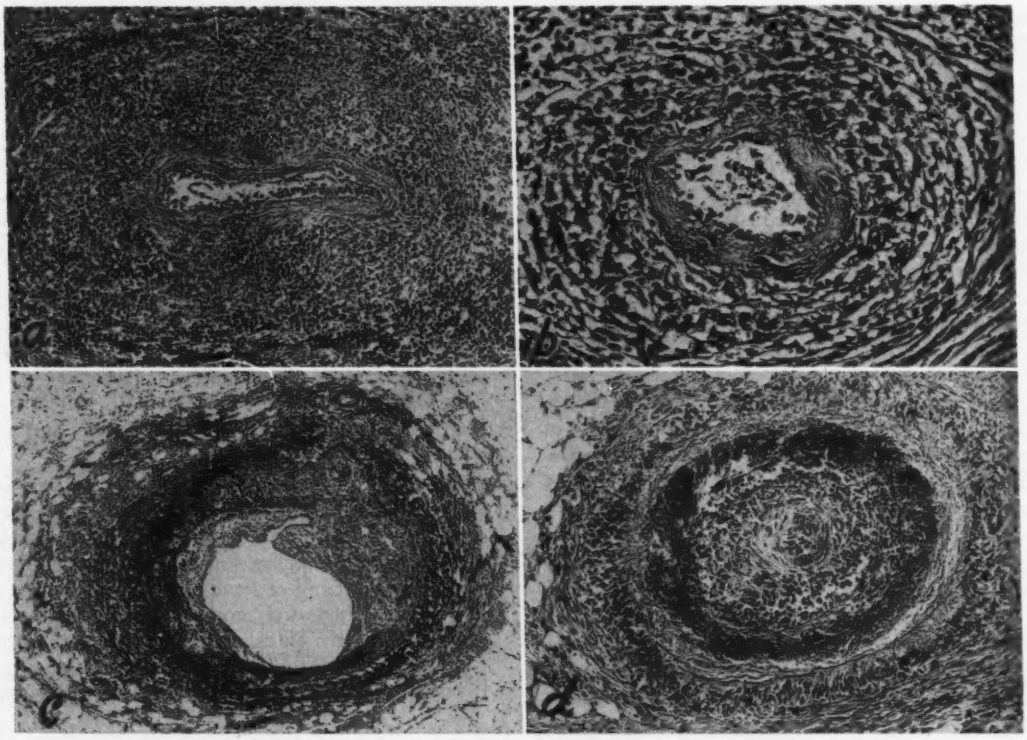

Fig. 1a. Arteriole from a section of liver. The vessel wall has been destroyed by necrosis and a cellular exudate of polymorphonuclear leukocytes extends to the inner media. Fibrinoid necrosis has destroyed the intima. (hematoxylin and eosin; reduced from x 140)

b. Arteriole from the adrenal capsule. A broad band of fibrinoid necrosis involves and replaces the intima and subjacent media. An edematous granulation tissue involves the media and adventitia. (hematoxylin and eosin; reduced from x 245)

c. Small artery from a section of tricuspid valve. Necrosis and an inflammatory reaction involve all layers. Note the marked intimal proliferation and disruption of the internal elastic lamina. (Verhoeff's elastic tissue stain with van Gieson counterstain; reduced from x 34)

d. Arteriole from a section of sciatic nerve. Note the three distinct zones which bear a resemblance to the histologic features of the rheumatoid subcutaneous nodule: (1) in the center is intimal proliferation and cellular reaction; (2) in the outer media is a band of "fibrinoid necrosis" surrounded by (3) radially arranged fibroblasts in palisade formation. (hematoxylin and eosin; reduced from x 130)

all 3 it was an early manifestation of the vascular disease. Also in all 3, histologic study gave ample evidence of widespread involvement of vessels in sections of peripheral nerves.

Careful analysis of the clinical records was done to compare the 4 patients who had disseminated necrotizing arteritis with the remainder of the patients in this series. No significant difference between the 2 groups could be found in regard to the onset, manifestations, severity, or progression of the arthritis, or to the treatment received, except for the administration of cortisone and evidences of hypercortisonism in the group with arteritis.

It is true that cases of necrotizing arteritis associated with rheumatoid arthritis were reported before the era of adrenal steroid therapy. It is also true that in reviewing the literature one finds descriptions of various degrees of "arteritis" occurring in patients with rheumatoid arthritis who have never received adrenal steroids. However, these cases are in the minority, and the overwhelming majority of cases reported of patients with rheumatoid arthritis who developed extensive diffuse acute necrotizing arteritis have followed adrenal steroid therapy.[1, 2, 4, 6, 7, 12, 13] It may be, as some have suggested, that the patients receiving cortisone were selected because of more severe manifestations and thus were more prone to develop complications from the onset. This, however, fails to explain the absence of necrotizing vascular lesions in the patients in our series from the precortisone era when all cases, severe and mild, received essentially the same type of therapy. It seems difficult to reconcile the apparent incrimination of cortisone as a causal agent in the production of necrotizing arteritis in this series and in other reports with the vast array of experimental and clinical evidence of the ability of adrenocortical hormones to heal or inhibit the production of these lesions. The answer to this apparent paradox may be in the relative disproportion between supply of and need for adrenal cortical hormones produced in these patients. As Bunim has stated, "While there are obvious statistical pitfalls in drawing firm conclusions about the role of steroid therapy in potentiating the development of the vascular lesions, there certainly is room for serious concern."[12]

Others have raised the question of whether the patients manifesting these "lupus-like" or "polyarteritis-like" syndromes did not actually have these diseases from the onset, instead of rheumatoid arthritis. This is a possibility. However, these reactions have been seen in patients whose course to that time had been typical of classic rheumatoid arthritis for many years; and after correction of the chronic hormonal overdosage, many have reverted to a course more typical of rheumatoid arthritis than of lupus erythematosus or polyarteritis.

In any event, current evidence seems to indicate that in certain susceptible patients with rheumatoid arthritis, chronic hormonal overdosage may result in the appearance of a mesenchymal reaction simulating lupus erythematosus or polyarteritis nodosa.

PREVENTION AND TREATMENT

Because of the potential seriousness of the manifestations of hypercortisonism, prevention of this condition is important. Proper selection of patients for steroid therapy and understanding and acceptance by both the patient and the physician of safe dosage levels are imperative. Initiation of steroid therapy in lower dosages and attainment of suppression of symptoms gradually is preferable to initial massive doses with quick and total suppression of all symptoms. Quick suppression of all symptoms plus the euphoria and stimulation associated with these excessive doses make it difficult for the patient to accept less complete relief of symptoms later on.

Women and particularly postmenopausal women are more susceptible to chronic hypercortisonism than other patients. They seem less able to tolerate high dosages over prolonged periods without developing this syndrome. Usually the special features of hypercortisonism described earlier do not become evident in less than 2 months of therapy with doses of 75 to 100 mg. of cortisone daily. Smaller but excessive doses may give rise to symptoms after longer periods of time, however. Currently recognized limits of dosage needed to avoid hypercortisonism were recently suggested by Slocumb and coworkers (table 1).

Treatment of hypercortisonism once it has appeared may be long and difficult. The basis of treatment is the gradual but steady reduction in dosage and, if possible, complete withdrawal of steroid therapy. The importance of small decrements in the success of any withdrawal program cannot be emphasized too strongly. Initial reductions may be larger,

TABLE 1.—*Maximal Daily Doses for Long Term Hormonal Therapy in Rheumatoid Arthritis.*

Group	Hydrocortisone (mg.)	Prednisone (mg.)
Children	<7.5	<2
Adolescents	7.5 - 10	2 - 3
Postmenopausal women	15 - 25	3.5 - 5
Premenopausal women	25 - 30	5 - 6.5
Adult men	30 - 40	6.5 - 9

After Slocumb, C. H.; Polley, H. F.; Ward, L. E.; and Hench, P. S.: Diagnosis, Treatment, and Prevention of Chronic Hypercortisonism in Patients with Rheumatoid Arthritis, Ann. Int. Med. 46:86-101 (Jan.) 1957.

but subsequent decrements should be in the range of .5 to 1 mg. of prednisone or equivalent doses of other steroids. The interval between reductions may vary from a few days to several weeks. Larger or more rapid reductions may produce actual flare-ups in the disease process or a "mesenchymal reaction" as described previously. Either of these may be not only discomforting, but dangerous to the patient. If such occur, further reduction may have to be delayed for long periods, even though undesirable effects are still present. The steroid dose should be divided into 6 or 8 hour intervals with as near even distribution of the dose as possible, unless variation in symptom pattern make unequal division more practical. It is essential that the patient realize that recovery from the undesirable effects of the special features of hypercortisonism mentioned may take a long period of time and that periods of considerable discomfort may occur.

Most patients are anxious to be relieved of the undesirable side effects of steroid therapy, but they are afraid that any lowering of their dosage will be impossible without depriving them of all antirheumatic relief. This is usually due to the fact that prior attempts at reduction of dosage have been made using too large a decrement in too short a period of time. Actually these people will feel more comfortable after proper reduction of dosage has reduced the symptoms due to hormonal overdosage. Needless to say a cooperative patient with insight into the aims of the program is also essential.

Other general measures are of help. Extra rest is extremely important. In many severe cases almost complete bed rest is necessary at the beginning of the withdrawal program. Full salicylate dosage and a conservative physical therapy program as tolerated should be continued. The use of tranquilizers such as meprobamate and chlorpromazine may be of help. Although some authors advocate the use of regular injections of Corticotropin, its use has not shortened the period of time necessary for recovery from chronic hypercortisonism in my experience. Treatment of the severe "mesenchymal reactions" is difficult. In the presence of a clinical picture similar to lupus erythematosus or polyarteritis, certainly further steroid withdrawal should not be attempted. On the contrary, large temporary increases in dosage may be necessary in order to control symptoms. Later after clinical improvement, more gradual withdrawal may be resumed.

SUMMARY

Patients with rheumatoid arthritis treated with excessive doses of adrenal steroids may develop a syndrome in addition to the usual manifestations of Cushing's syndrome. This is characterized by a triad of cyclic mood changes, fatigability, and musculoskeletal aching. Furthermore, these patients seem susceptible to so-called "mesenchymal reactions" which may simulate lupus erythematosus, a flare of rheumatoid arthritis, or periarteritis nodosa. Treatment of these special features of hypercortisonism consists in the steady, gradual reduction of dosage in small decrements according to the patient's reaction to the withdrawal.

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Study Shows Majority Satisfied with Insurance

Seven out of every 10 persons with health insurance express complete satisfaction with it, reported the Health Information Foundation recently. This finding was based on a survey made by the National Opinion Research Center of the University of Chicago of a cross section of almost 2,400 persons, as well as approximately 500 family physicians.

The foundation's report is limited to what the public and family doctors said about voluntary health insurance. Even among the minority without health insurance, three out of five thought that health insurance was a good idea. Suggestions made for improving existing coverage were that insurance should cover more services, such as doctors' home and office visits, and a higher proportion of the total bills for presently covered hospital surgical services. One person in six complained that premiums were too high.

It Might Be Histoplasmosis

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HISTOPLASMOSIS is a disease which many or perhaps most of us tend to look upon as simply a pulmonary disease which is endemic in the Mississippi Valley and which produces roentgenologic changes in the lungs that simulate those produced by tuberculosis. But there is more, much more, to this frequently fatal disease. Among a number of other possible manifestations, there can be and often are primary or secondary granulomatous or neoplastic appearing nodules or ulcerations of the skin and/or mucous membranes. Dermatologists and otorhinolaryngologists in particular, and physicians in general, should keep alert to the possibility that such lesions might be histoplasmosis.

Dr. Myles P. Moursund and his co-author, both dermatologists in private practice, presented this paper for the Texas Dermatological Society, April 21, 1958, in Houston.

Mucocutaneous manifestations, especially granulomatous or neoplastic appearing nodules or ulcerative lesions in the buccal cavity, on or near the lips, in the nasal cavity, or in the pharynx, may be signs of histoplasmosis. A biopsy supplemented by culture will establish the diagnosis. Amphotericin B is the treatment of choice. A case of histoplasmosis is reported.

Our recent discovery of a case of mucocutaneous histoplasmosis stirred us to make a survey of all dermatologists, several otorhinolaryngologists and several pathologists in Texas to estimate the incidence of histoplasmosis in this state and further to estimate the frequency of mucocutaneous lesions of the disease. Sixty-nine replies to 88 questionnaires gave us the positive results charted in figure 1, which shows the distribution of 22 cases seen in Texas by this group of doctors. These survey results will be discussed in a little more detail later.

Mullins[12] reported that the records of John Sealy Hospital, Galveston, revealed that the diagnosis of histoplasmosis had been made 31 times in that hospital. A check of the disease index records of all hospitals in Houston revealed that the diagnosis of histoplasmosis had been made 46 times in these hospitals. These statistics obviously do not reflect the complete incidence of histoplasmosis in Texas, but they point toward the conclusion that there have been an appreciable number of cases seen in widely scattered locations, seemingly concentrated in the eastern half of the state. Figure 1 also shows that, within a short distance from Texas, we had replies from 4 dermatologists in New Orleans who reported each having seen from 3 to 20 cases of histoplasmosis, with as many as 10 to 15 cases having had mucocutaneous lesions. This, just as an example, suggests the higher percentage of cases occurring in the endemic area of the Mississippi Valley.

There have been a number of excellent papers on the subject of histoplasmosis. It is not the purpose of this paper to fill numerous pages with a full review of the literature, but for those who wish more complete details, we refer to the bibliography.[1, 6, 10, 11, 13] A case report exemplifying probable primary mucocutaneous histoplasmosis will be presented, our survey findings will be further explained, and then pertinent abstracts from the literature will be summarized.

REPORT OF CASE

A 76 year old white truck farmer of La Porte was admitted to a Houston hospital January 29, 1957, complaining of painful sores at the corners of his mouth. These had appeared about 9 weeks before. A few days after the onset of these lesions, he noticed a tiny nodule in the midline of the upper gum. This soon became eroded. Movement of the lips caused considerable pain, and saliva drooled freely from the lip commissures. Vitamin therapy and topical applications prescribed by the referring physician had been of no benefit.

Fig. 1. Results of partial survey of Texas doctors on active histoplasmosis cases seen.

On direct questioning, the patient admitted to spells of nonproductive coughing, occurring usually in the morning, for the past 1 or 2 years.

The ulcers at the labial commissures appeared whitish and macerated with slightly undermined edges and with smooth bases covered by a delicate gray coagulum. They were indurated. Patches of tiny erosions were present in the midline of the upper gum, on the right hard palate area, and at the base of the uvula on the left (fig. 2).

A roentgenogram of the chest showed fibrotic changes in the apex of the right lung, considered most likely to be the result of old tuberculous disease. A 4 cm. round density was seen in the anterior part of the left upper lobe. This was felt to be either metastatic carcinoma or a primary carcinoma of the lung. No mention was made of anything suggesting histoplasmosis of the lungs, but in our opinion, only an exploratory thoracotomy could have decided whether the large density in the left upper lobe was carcinoma or a solitary nodule of histoplasmosis. Blood count and urinalysis were normal, and a Kline test was negative. Culture of the lip and buccal cavity lesions at the hospital yielded a yeastlike growth considered to be Saccharomyces. Biopsies of both lip and mouth lesions showed a granulomatous infiltrate with large histocytes containing organisms, readily identifiable as Histoplasma capsulatum (fig. 3).

The patient refused to undergo an exploratory thoracotomy for investigation of the lesion in the left lung and he elected to seek further therapy elsewhere.

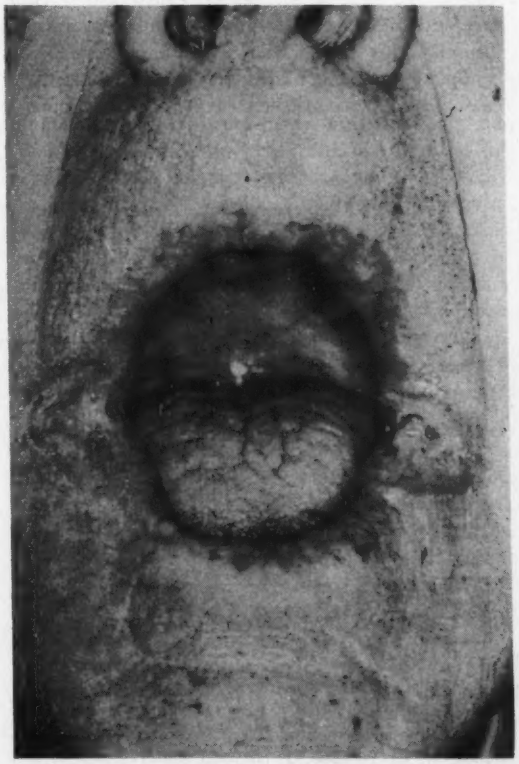

Fig. 2. Painful, whitish, macerated ulcers of the labial commissures before treatment.

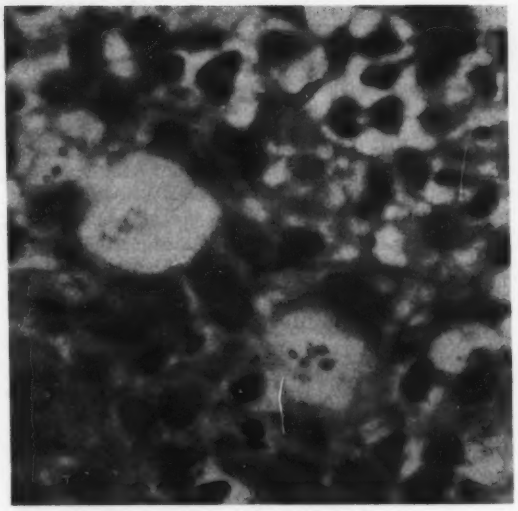

Fig. 3. Histoplasma capsulatum in lip biopsy tissue.

The patient was first seen at our office on February 4, 1957, and presented the lesions described previously. A review of systems and physical examination were otherwise negative. A check with the pathology department of the hospital revealed their finding of H. capsulatum in the biopsy specimens of our patient. Another culture on Sabouraud's glucose agar was taken from the lip and buccal cavity lesions. The patient was given Triple Sulfa tablets in dosage of 8 tablets per day, and he was also given sodium caprylate (Naprylate) 10 per cent solution for a mouthwash, three times a day. One week later the lesions showed considerable healing; there was further improvement when he was seen on March 11, and all of the lesions appeared completely healed by April 8, approximately 1½ months after treatment was initiated. Treatment was discontinued at this time and check-ups of the patient on

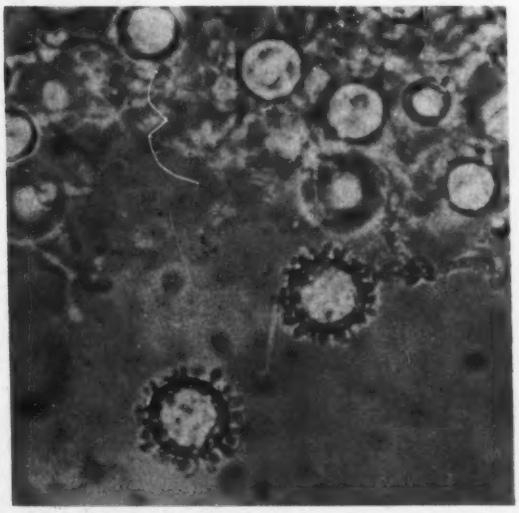

Fig. 4. Culture mount (room temperature culture) showing tuberculate chlamydospores of H. capsulatum.

July 8 and December 10 revealed no evidence of activity of his histoplasmosis. As is to be expected, there was no evidence of growth on his culture for about 1 month after it was taken (a small white tuft in early March) and it was not until the end of March that tuberculate chlamydospores, which definitely identify H. capsulatum, could be found in the culture mounts (fig. 4).

Having not been done previously, a histoplasmin skin test (with 1:10,000 dilution) and a histoplasmosis complement fixation test were done on December 10, and both tests were negative. A repeat roentgenogram of the chest made in December showed essentially the same findings as the one taken in January except that the tumor mass (considered probably malignant) in the upper lobe of the left lung was more irregular in shape and was at least twice as large. The roentgenologist stated that he could see nothing in the film that would suggest histoplasmosis past or present. This chest film report, plus the negative report of the previous film in January, plus the lack of signs or symptoms suggestive of other foci of internal histoplasmosis seem to confirm or at least support our impression that this patient did have primary mucocutaneous histoplasmosis.

DISCUSSION

Although histoplasmosis is known to be worldwide in distribution, it is considered to be endemic in only a rather limited portion of the United States, consisting of southern Illinois, Indiana, Iowa, Kansas, Kentucky, Missouri, Ohio, and Tennessee.

As stated previously, our survey reveals that Texas also comes in for its share of histoplasmosis cases. Reports from 69 doctors show that 15 dermatologists, 1 otorhinolaryngologist, and 2 pathologists have seen a total of approximately 34 patients (22 in Texas) with proven histoplasmosis. We say "approximately" because in spite of our efforts to avoid it, a few of this number may still be duplications. Also the case in Brownsville, considered to have been histoplasmosis of the nasal mucous membrane, we believe to have been a case of rhinosporidiosis, according to our interpretation of the biopsy slides which were kindly loaned to us. Of the 22 patients seen in Texas, 17 were considered to have contracted their histoplasmosis in this state. Another patient may have developed histoplasmosis in Houston, but possibly brought it with him from South America. Four patients seen in Army or Veterans Administration hospitals in Texas probably contracted histoplasmosis elsewhere and were sent to these hospitals for treatment. Of the 34 total cases observed, 22 had disseminated histoplasmosis when first seen, and 6 of these had mucocutaneous lesions. Twelve patients were considered to have had primary mucocutaneous infection initially, but 5 of these disseminated later and died. Of the reports that revealed the location of the primary mucosal or cutaneous lesions, there were 3 in the oral cavity, 2 oral cavity and lip, 1 lips, 1 nasolabial, 1 nasal septum, 1 larynx, 1 cutaneous (site not specified), 1 lips and penis, and 1 involving the lips and perianal area.

Figure 5, based on a histoplasmin test survey by the United States Public Health Service, shows even more vividly the histoplasmosis potential in Texas with again a heavy concentration in East Texas but also a fairly heavy concentration in Southwest Texas.

A report obtained from Dr. George G. Browning[8] of the Texas State Department of Health revealed that histoplasmosis does kill people in Texas. From 1949 through 1956 histoplasmosis was reported as the cause of death in from 1 to 5 persons each year.

It is difficult or perhaps impossible to determine from a large amount of literature reviewed, just how many cases of mucocutaneous histoplasmosis have been reported. There is undoubtedly some duplication in at least the reviewed cases. Miller and associates[10] reviewed 78 cases of histoplasmosis discussed by Parsons and Zarafonetis,[13] found 17 more cases in the literature, and reported a case of their own. In these 96 cases of histoplasmosis, cutaneous or mucocutaneous lesions were observed in 45 (more or less 50 per cent of the cases). Curtis and Grekin[6] reviewed 21 cases (12 with definite mucocutaneous histoplasmosis and 3 with possible mucocutaneous histoplasmosis) and presented 3 new mucocutaneous cases. Thomas and Mitchell[16] reported a disseminated

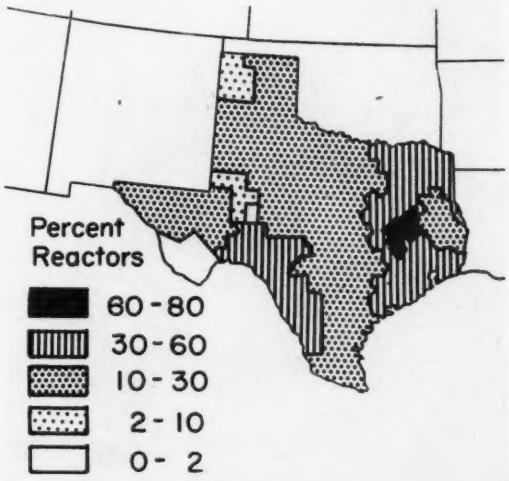

Fig. 5. Histoplasmin sensitivity in Texas, revealed in a survey performed 1945-1951 by the United States Public Health Service. (Adapted by Furculow, M. L., from Manos, N. E.; Ferebee, S. H.; and Kerschbaum, W. F.: Geographic Variation in Prevalence of Histoplasmin Sensitivity, Dis. Chest 29: 649-668 [June] 1956.)

case with generalized cutaneous nodules. In 1948 Weed and Parkhill[17] reviewed 73 cases, 24 of which included oral lesions, and they presented 4 new cases with oral or pharyngeal lesions. Finally in 1957 Baum and associates[1] presented 3 cases of mucocutaneous histoplasmosis, and Plotnick and Cerri[14] presented 1 more case with oral lesions. These, with our case and the 17 others with mucocutaneous lesions seen by our Texas associates, make a total of approximately 87 patients with mucocutaneous histoplasmosis reported so far.

The reviewed literature on histoplasmosis leads one to believe that it is almost, if not completely, a unanimous opinion that the primary infection is always internal, usually in the lungs, and that this infection either heals spontaneously or becomes disseminated throughout the body by embolism or by direct extension or by secondary inoculation. For example, Silverman and associates[15] stated the aforementioned opinion and denied the possibility that the skin or mucous membranes are primarily infected because of the rarity in which regional lymph nodes are involved. The disease is also considered, at least in the majority of reported cases, to be invariably fatal. Baum and associates,[1] on the other hand, took the stand that there are 3 general types of cases of histoplasmosis with mucocutaneous lesions as follows: (1) Fulminating active disseminating cases with associated skin or mucosal lesions. (2) Slowly progressive disseminating cases with skin and mucosal lesions as the only manifestation, or one of the few manifestations, but with a gradual downhill course, in which the patients, unless killed otherwise, eventually die of histoplasmosis. (3) Cases with skin or mucosal lesions as the only demonstrable signs of the disease in which the patients get well and stay that way. This evaluation or classification of mucocutaneous histoplasmosis cases seems the most logical one to consider. But even Baum and associates[1] expressed the belief that there is no true primary mucocutaneous histoplasmosis.

We were unable to review in detail the 34 histoplasmosis cases seen by Texas physicians, but from what information we received, it appears that 12 were primary mucocutaneous cases. Five of these patients later died of disseminated histoplasmosis; so perhaps these 5, or at least some of them, were systemic or internal to begin with. One of the 12, our own case, we believe was definitely primary mucocutaneous histoplasmosis. In our survey Burks and Hennington[4] of New Orleans, respectively reported 3 and 1 primary mucocutaneous cases. So, we estimate that at least 10 to 12 cases of primary mucocutaneous histoplasmosis have now been reported or observed.

DIAGNOSIS

The types of mucocutaneous lesions due to, or associated with, histoplasmosis as listed by Miller and associates[10] are ulcerations and granulomas of the oral mucosa, tongue, or larynx; perforated nasal septum; plaque on tongue; small discrete dome shaped nodules on larynx, vocal cords, epiglottis, and posterior tongue with ulceration and destruction of involved organ later; cutaneous papules, plaques, and punched out ulcers; purpuric lesions, especially in infants; abscesses and furunculoid and impetiginized areas; and localized or generalized dermatitis including scaling hands and feet, verrucous dermatitis, and vegetative and ulcerating lesions. From this we can see that the mucocutaneous lesions of histoplasmosis are protean in appearance. These lesions are not sufficiently characteristic to permit one to make a definite diagnosis from the clinical appearance alone, but the causative organisms are demonstrated readily by microscopic examination and culture of biopsy specimens from a surprisingly large number and variety of lesions.

An idea of the confusion in the diagnosis of histoplasmosis that existed at least prior to 1948 is illustrated by a statement made by Weed and Parkhill[17] in their review of 73 cases. Though a few of these cases had been recognized early as histoplasmosis, the large majority of cases had initial clinical diagnoses such as syphilis (primary or tertiary), impetigo, breast abscesses, aleukemic leukemia, trench mouth, typhoid fever, Addison's disease, splenic anemia, suppurative arthritis, gallbladder disease, gastric ulcer, or carcinoma. Also much confusion existed in the interpretation of the biopsies in these cases. Some of the pathologic diagnoses were chronic adenitis, indeterminate granuloma, blastomycosis, lymphoblastoma, Hodgkin's disease, leishmaniasis, kala azar, carcinoma, and tuberculosis. Of course, most of these clinical and pathologic diagnoses would have to be considered in the differential diagnosis, but "it might be histoplasmosis."

Taking another note from Weed and Parkhill,[17] we agree and wish to emphasize that histoplasmosis may be recognized readily and proved with a minimal amount of equipment and trained personnel, using techniques that are generally available. A biopsy supplemented by a culture are the only essential tests needed. It would be well to retain a small portion of the biopsy tissue so that additional bacteriologic investigation may be accomplished should the histologic examination not reveal a neoplastic or identifiable granulomatous process and should the original culture fail to grow out or be so contaminated as to be unidentifiable. An important point in regard to cultures that was emphasized by Thomas and Mitchell[16] is that one must realize and remember that growth of H. capsulatum, both at incubator

and at room temperatures, is very slow (12 to 18 days for growth to appear). The common practice of too rapid discarding of cultures obviously may preclude a diagnosis of histoplasmosis by this means. In our case, the diagnosis had already been established by biopsy at the hospital, but the culture there was reported as "yeastlike growth, apparently of Saccharomyces." This growth occurred on blood agar at 37 C. It could have been the yeast phase of H. capsulatum, and no doubt this and the room temperature Sabouraud's glucose agar culture were discarded promptly before any characteristic chlamydospores could develop and be demonstrated. Our own culture required 1 month before any growth could be seen, and then it was about another month before the tuberculate chlamydospores could be demonstrated in culture mounts.

Since, in the systemic form of the disease, the lungs are almost invariably involved, serial roentgenograms of the chest should reveal a diffuse pneumonitis or eventually reveal the more or less characteristic miliary calcification of healed histoplasmosis.

Furcolow and associates[8] offered a word of caution in a study of laboratory infections with H. capsulatum. The study showed that work with this organism constitutes a hazard similar to that of working with Coccidioides immitis.

TREATMENT

Treatment of histoplasmosis can be summed up rather briefly. To date there is still no specific treatment for histoplasmosis. Brasher and Furcolow[2, 7] in 1955 reported treatment of 21 cases of chronic bilateral cavitary pulmonary histoplasmosis. Of these, 12 received 8-dimethyl-amino-ethyl fencholate (MDR-112) (William S. Merrell); 3 received 2 hydroxy-stilbamidine (William S. Merrell); 3 received aminostilbamidine; 1 was treated with nystatin (Squibb); 1 with testosterone; and 1 with Antabuse (tetraethyl-X) (Ayerst). Patients were treated for 30 to 60 days with no toxic effects, but also with no significant therapeutic effect. Brasher and Furcolow concluded that a trial of some of these drugs for 90 to 180 days might prove worth while. Mayer and associates[9] thought that they had demonstrated conclusively both in vitro and in mice experiments that sulfa drugs, particularly sulfadiazine and sulfapyridine, not only exert prophylactic action but are definitely curative of histoplasmosis. However, case 1 of Baum and associates[1] received intensive oral therapy with both nystatin and sulfonamides and died in less than a month. Campbell[5] using a new antibiotic, 1968 (Nepera), by injection in mice, found that this drug definitely increased survival time and was nontoxic for mice in the concentrations employed, and she concluded that it warrants a trial in man. Dr. Furcolow in a talk at the Texas Tuberculosis Association meeting in Houston on March 29, 1958, revealed that within the past 2 years good results had been obtained in the treatment of a limited number of critically ill histoplasmosis patients by the use of intravenous amphotericin B, an antifungal antibiotic derived from a new species of Streptomyces.

For localized mucocutaneous lesions the latest reported successful treatment was that of Plotnick and Cerri[14] in which buccal lesions were cured by local injections of nystatin solution. In the same patient, 1 lesion on the lower lip apparently was cured by excision biopsy and 2 lesions on the upper lip were cured by excision, with no reported use of nystatin in these labial lesions. In the reviewed literature cures and failures have been reported with excision, radium therapy, and roentgen-ray therapy. Our case either healed spontaneously or appeared to respond to a combination of, or possibly to one or the other of, Triple Sulfa tablets and sodium caprylate mouthwash.

SUMMARY

A survey questionnaire to all dermatologists, several otorhinolaryngologists and several pathologists in Texas, concerning the incidence of histoplasmosis in this state resulted in 69 replies to 88 cards sent out. The replies revealed that 15 dermatologists, 1 otorhinolaryngologist, and 2 pathologists had seen a total of approximately 34 proven cases of histoplasmosis. The word "approximately" is used because possibly a few of these cases may have been duplications. Of the 34 patients, 22 were seen in Texas and 18 were considered to have contracted their disease in Texas with seemingly a concentration of cases in East Texas. Twenty-two of the 34 patients had disseminated histoplasmosis when first seen and 6 of these had mucocutaneous lesions. Twelve patients were considered to have had primary mucocutaneous infection initially, but 5 of these later had disseminated disease and died. A histoplasmin sensitivity test survey done by the United States Public Health Service in the period 1945-1951 revealed that about 30 per cent of all Texans were histoplasmin positive, Southwest Texans were 30 to 60 per cent positive, and East Texans were 30 to 80 per cent positive. Records of the Texas State Department of Health show that histoplasmosis has been reported as the cause of death in from 1 to 5 persons each year since 1949. So, there definitely is histoplasmosis in Texas.

Reviewing the literature and adding the cases reported to us by Texas doctors, we arrive at a total of approximately 87 patients now reported as having had mucocutaneous lesions of histoplasmosis. Also

it appears that 10 to 12 of these patients had primary mucocutaneous histoplasmosis.

Although the mucocutaneous manifestations of histoplasmosis, as described in the literature, are protean in appearance, the most frequent and more or less characteristic lesions are granulomatous or neoplastic appearing nodules or ulcerative lesions. Any such lesions seen, particularly in the buccal cavity or on or around the lips or in the nasal cavity or in the pharynx, should receive thorough evaluation in the way of differential diagnosis. Similar evaluation should be given to such lesions occurring anywhere on the skin, in the larynx or on the anogenital area. "It might be histoplasmosis."

A biopsy supplemented by culture is all that is necessary to establish a diagnosis of histoplasmosis. Histoplasma capsulatum is slow growing; therefore, cultures should be retained and examined periodically for at least 1 month.

There is no specific therapy for histoplasmosis, but research studies to find a specific therapeutic agent are continuing. Amphotericin B given intravenously is the treatment of choice at the present time.

A case of Texas style mucocutaneous histoplasmosis is presented.

We are indebted to all of the doctors who contributed to our survey on histoplasmosis; to H. S. Gallagher, M.D., of M. D. Anderson Hospital and Tumor Institute, Houston, who supplied us with some of our clinical and photographic material; and to Michael L. Furcolow, M.D., Kansas City, Kan., for permission to use and reproduce his map "Histoplasmin Sensitivity in Texas."

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ABSTRACT OF DISCUSSION

DR. JOHN M. KNOX, Houston: It might be well to discuss some of the immunological aspects of deep fungus diseases. Although the following comments are particularly pertinent in regard to histoplasmosis, they also apply to most of the deep fungus diseases.

A positive histoplasmin skin test indicates that the patient has had an encounter with Histoplasma capsulatum and has developed a sensitivity to the organism. Although sensitivity and immunity do not necessarily parallel one another, a positive skin test often indicates a relative degree of tissue resistance to the organism. The individual in his initial contact with the organism may or may not have symptoms, and even when present, the clinical symptoms are rarely diagnosed as histoplasmosis in this initial phase. Also, the patient may or may not develop the characteristic calcification in the lungs.

The complement fixation test is dependent upon the presence of a protein or protein-like substance containing nitrogen. When positive, this test roughly indicates the amount of tissue involved by the disease process, and a strongly positive complement fixation test indicates widespread dissemination of the disease. In general, the complement fixation test will become less positive as a patient improves or will increase in titer if the disease progresses.

The precipitin test depends upon the presence of specific polysaccharides. This test becomes positive early in the disease process and then progresses to negativity in spite of the fact that the disease may be increasing in extent. A positive precipitin test, therefore, indicates that the disease is relatively recent in onset.

In summary, in histoplasmosis and similar deep fungus diseases it is worth while to correlate the skin test, complement fixation test, and precipitin test for a better understanding of the patient and the disease. A positive skin test and a negative or low titer complement fixation test indicate a relatively good prognosis, whereas on the other hand a negative skin test and a high titer complement fixation test indicate a poor prognosis. The precipitin test gives an indication as to the duration of the disease.

Afibrinogenemia

A Review

GILBERT LANDIS, M.D.

El Paso, Texas

IN 1901 DeLee reported the failure of the blood to clot in a patient with abruptio placenta, first drawing attention to this condition. DeLee described it as a temporary hemophilia. In 1915 Williams made a similar observation and likened the hemorrhagic lesions to those seen after poisoning by certain varieties of snakes. He suggested that a toxic substance circulating in the blood led to changes in the vessels that permitted bleeding into the tissues. In 1922 Wilson hypothecated that in abruptio placenta a hemorrhagic toxin was liberated from the placenta, leading to further placental separation. He proposed that absorption of this toxin resulted in a generalized hemorrhagic tendency. In 1936 Dieckman found depressed fibrinogen levels were present with incoagulable blood in abruptio placenta. Dieckman suggested that the reduction in fibrinogen level was due to mobilization of fibrin at the site of the placental separation. Maloney, in 1944, identified fibrinogen depression as a causative factor of hemorrhage of incoagulable blood in patients with an intrauterine retention of a dead fetus and abruptio placenta. He attributed the afibrinogenemia to the presence of a fibrinolysin. In 1944 Steiner and Lushbaugh described the amniotic fluid syndrome, and the hemorrhage of incoagulable blood, which occurs should the patient survive a sufficient period of time.

The incidence of clinical hypofibrinogenemia or afibrinogenemia in Nassau County, N. Y., in 1953 to 1954, was 1:3,148 cases, with a maternal mortality rate of 10 per cent.

CONDITIONS OF OCCURRENCE

A large variety of clinical conditions is associated with fibrinogen depression. Among these are abruptio placenta, intrauterine retention of a dead fetus, amniotic fluid infusion or amniotic fluid embolism, convulsive eclampsia, delayed postpartum hemorrhage, surgical trauma, generalized Shwartzman phenomenon, bilateral cortical necrosis of the kidneys, pituitary necrosis or Sheehan's disease, forceps trauma, hydatidiform mole with hemorrhage and toxemia, bacterial infections, and unusually intense muscular contraction as occurs in obstructed or unusually strong labor.

PATHOLOGIC MECHANISM AND ETIOLOGY

The pathologic mechanism whereby tissue thromboplastin activates the fibrinogen-fibrin conversion process has a dual symptomotology:

1. The initial impact causes obstetric shock from massive intravascular fibrin embolism.
2. There is hemorrhage of incoagulable blood caused by severe fibrin depletion.

In the experimental animal, injection of thromboplastin intravenously causes depression of circulating plasma fibrinogen with the speed and degree of a chemical reaction. The reaction is reversible with fibrinogen recovery occurring almost with equal promptness. The liver has a tremendous capacity of regenerating fibrinogen. It is possible to correlate the relative fibrinogen values with incoagulable blood, blood clot stability, obstetric shock, critical plasma fibrinogen level, and effectiveness of therapy. Various theories have been proposed to explain the depression of fibrinogen under certain clinical circumstances. They include the mobilization of circulating plasma fibrinogen to the site of trauma as a function of the body's protective mechanism. A second theory is the presence of abnormal fibrinolysis also related to the body's protective mechanisms. However, the most widely accepted theory is hyperthromboplastinemia with its defibrinating influence. This theory assumes thromboplastin to be the initiating influence. When thromboplastin is introduced into the circulating blood, in the presence of other essential blood factors, prothrombin is converted to thrombin, which in turn causes fibrinogen to be converted to fibrin. The presence of fibrin in the circulating blood results in a disseminated embolization with occlusion of the terminal arterial radicals of the capillary bed. The depressed plasma fibrinogen level of the circulating blood reflects the extent of the defibrination process. Defibrination of circulating blood may be a complication of any tissue catabolic activity of greater than ordinary magnitude. This is particularly true when the process involves muscle. Unusually intense or pathologic contraction of individual muscles may be sufficiently catabolic to produce defibrination. Normal labor and delivery do not alter plasma fibrinogen concentration significantly. The mean average in pregnancy and puerperium is 315 mg. of fibrinogen per 100 cc. of plasma, whereas the mean average in a normal nonpregnant female is 267 mg. per 100 cc. of plasma. The critical level, below which clinical symptoms develop, is in the range of 150 to 90 mg. of fibrinogen per 100 cc. of plasma.

Abruptio Placenta.—Abruptio placenta almost always presents to a degree depression of fibrinogen. This varies from total afibrinogenemia to minor degrees of hypofibrinogenemia, with which there is a self limitation of the fibrinogen depressing process. It has been proposed that the retroplacental hematoma mobilizes fibrinogen which is converted to fibrin. However, there are apparently other factors involved. Myometrial injury appears to be highly important in the degree of defibrination, which in turn is directly proportional to the severity of the placental abruption. As long as there is continuing myometrial damage, the process of defibrination generally progresses. In mild placental separations, however, the defibrination process may be self limiting. Experimental studies in the dog by Foster and Whipple show that dogs regenerate fibrinogen at the approximate rate of 38 mg. per 100 cc. of blood per hour. Hodgkinson and his group found that in abruptio placenta, the fibrinogen recovery rate averaged 20.40 mg. per 100 cc. of plasma per hour following delivery, thus suggesting that the hemorrhage of incoagulable blood from an untreated critical fibrinogen depression could be expected to continue from 2 to 4 hours following delivery. The termination of pregnancy in an abruptio placenta abruptly reverses the downward fibrinogen trend.

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Clinical afibrinogenemia may be associated with abruptio placenta, retention of a dead fetus, amniotic fluid infusion, and other conditions related to pregnancy. It may cause obstetric shock and hemorrhage of incoagulable blood. If hemorrhage is not promptly controlled, defibrination should be suspected and appropriate therapy instituted.

Placenta previa is probably not a thromboplastin inducing condition since the blood readily gains egress from the uterus and produces little or no myometrial damage.

Dead Fetus Syndrome.—The Rh factor is only of apparent etiologic importance; hence relationship to the dead fetus syndrome is incidental to the increased frequency with which intrauterine death occurs in maternal Rh isoimmunization. A prominent feature of the dead fetus syndrome is the chronicity of the fibrinogen depression. Hypofibrinogenemia may be apparent weeks before the onset of labor. Recovery begins with evacuation of the uterine contents. However, unusually low values may persist for several weeks after delivery. Neither maternal trauma nor abnormal myometrial contractions are apparently related to the etiology in the dead fetus syndrome. The first suggestion of hypofibrinogenemia may be purpura. There is evidence to suggest that the chronic maternal absorption of amniotic fluid, which is rich in fetal catabolites, is the probable defibrinating influence. In the dead fetus syndrome, little change in fibrinogen levels can be expected in less than 4 weeks. In 90 per cent of the cases following death of the fetus in utero, spontaneous delivery can be anticipated within 4 weeks without risk of maternal disturbance of blood coagulation.

Amniotic Fluid Syndrome.—Amniotic fluid syndrome was first described in 1941 by Steiner and Lushbaugh. Hemorrhage of incoagulable blood will occur in this syndrome if the patient survives for a sufficient period of time. However, the symptoms of shock and cardiopulmonary insufficiency are so severe that most die before diagnostic studies or uncontrolled hemorrhage can result. It is difficult to correlate the severe obstetric shock and sudden death with a massive embolization resulting solely from particulate matter contained in amniotic fluid. It has been postulated that in addition to embolization from particulate matter of amniotic fluid, defibrination and massive fibrin embolism occur from the thromboplastin contained in amniotic fluid. However, amniotic fluid is relatively low in thromboplastin content. It is probable that a large volume is necessary to provoke the amniotic fluid syndrome. In addition, it is well known that chorionic villi frequently are found in the lungs of pregnant women without symptoms. It has been proposed that the term amniotic fluid infusion would be more appropriate than amniotic fluid embolism. It has been suggested also that the thromboplastin concentration in amniotic fluid is augmented from the prevailing circumstances attending the amniotic embolism, for instance, tetanic uterine contractions, meconium in amniotic fluid, advanced maternal age, large baby, multiparity, difficult operative delivery, and the presence of a dead fetus.

Surgical Trauma.—Consistent subclinical fibrinogen depression immediately following surgery occurs, persisting for as long as 9 hours. This is followed by a proportionately much greater hyperfibrinogen reaction, which persists for several days. The reaction varies directly with the degree of surgical trauma. The response apparently reflects fibrin utilization by the body's defense mechanism and utilization of the fibrinogen stimulates the further production by the liver. Others have suggested that the increased fibrinogen production results from the slow absorption of thromboplastin in low concentration.

Late Postpartum Period.—In one series of 32,155 deliveries, there were 32 cases of retained placental fragments causing late postpartum hemorrhage or an incidence of 1:1,005. The most frequent causes listed for late postpartum hemorrhage are (1) retained placental fragment, (2) uterine abnormality in the involution of the placental site, and (3) uterine abnormality in retention and separation of the decidua vera.

Products of conception, particularly when autolyzed, have a strong thromboplastic activity. The pathway of gaining entrance to maternal circulation is through the rupture of the basal decidual plate, allowing decidual debris, which is extremely rich in thromboplastin, to gain entrance to the maternal circulation. This apparently also occurs in abruptio placenta.

CLINICAL TYPES

Three types of fibrinogen depression can be described:

1. Hyperacute type, which occurs in amniotic fluid embolism with symptoms of pulmonary embolization and death from acute cardiopulmonary insufficiency. If the patient survives long enough, there will be manifest hemorrhagic symptoms resulting from a critical fibrinogen depression.

2. Acute type, such as occurs in abruptio placenta with mixed symptomotology. There is hemorrhage of incoagulable blood from a critical fibrinogen depression and obstetric shock from fibrin embolization.

3. Chronic type, which occurs in the dead fetus syndrome where there is long intrauterine retention of a dead fetus. This has an insidious onset and seldom is associated with the symptoms of embolization. The shock, if it occurs, is due to blood loss.

CLOT STABILITY

It has long been noted that in certain cases where the fibrinogen level is low, a clot will form and then subsequently be lysed. Fibrinolysin has been demonstrated in increased amounts. However, the instability of the clot in defibrination is related to the degree of fibrinogen depression. In afibrinogenemia, fibrinolysin is not a significant factor in etiology. Blood containing approximately 150 mg. of fibrinogen per 100 cc. of plasma serves as a dividing line between clot stability and instability. Between 150 and 100 mg. there is partial clot dissolution. Between 100 and 60 mg. a soft, fragile clot will form which promptly dissolves. When there is less than 60 mg. of fibrinogen per 100 cc. of plasma, there is complete failure of clotting. The clot instability is probably due to a combination of fibrinolysis and a weak clot due to the lack of an adequate amount of fibrinogen. Circumstances prevailing for the production of defibrination are known to result in increased fibrinolytic activity, for instance, fear, preoperative sedation, trauma, epinephrine, or muscular activity. Certain other blood factors are depressed but not critically; for instance, AC globulin, platelets, and prothrombin are usually depressed to a minor degree.

MANAGEMENT

In subclinical defibrination, interruption of the disease process is sufficient treatment. In clinical defibrination, the symptoms of embolization and obstetric shock, as well as traumatic and surgical shock, require supportive therapy. In cases of obstetric shock, one must beware of excessive transfusion, as transfusion is of no avail in obstetric shock without hemorrhage. In fact, minor degrees in reduction of blood volume would be beneficial in reducing to some extent the mechanical embarrassment of acute cardiopulmonary insufficiency resulting from massive embolization of the terminal pulmonary arterial radicals. The mechanism of dissolving the fibrin thrombi in circulating blood is not known. However, fibrinolytic activity and time are probably the most important therapeutic measures. Hemorrhagic defibrination requires restoration of blood volume and treatment for correction of the critical hypofibrinogenemia. Regeneration of fibrinogen will occur normally in the average patient within a few hours. The slowest recovery is in the dead fetus syndrome. As mentioned previously, dogs are able to regenerate fibrinogen at the average rate of 38 mg. per 100 cc. of blood per hour. Fibrinogen must be reserved for emergencies because of the inability to eliminate the virus of infectious hepatitis. A 500 cc. blood transfusion cannot be expected to raise the fibrinogen concentration more than 5 to 10 per cent. Fresh whole blood is better than bank blood. In fact, bank blood depresses fibrinogen levels to a maximum at 5 hours post-transfusion.

Amniotic fluid embolism usually occurs toward the end of the second stage of labor. If the patient's cervix is nearly completely dilated, amniotomy and rapid delivery is in order. If not, amniotomy and drainage of the amniotic fluid from above the level of the presenting part to relieve the intrauterine pressure is necessary. Cesarean section is unthinkable when the patient is in obstetric shock from the amniotic fluid embolism.

The management of abruptio placenta depends a good deal upon obstetric judgment. Either vaginal

or abdominal delivery may be resorted to after determination of the fibrinogen status of the patient's blood. If the patient's fibrinogen level is from 220 to 150 mg. per 100 cc. of plasma, interruption of pregnancy may be safely accomplished by either means. However, serial determinations are necessary to reveal the fibrinogen trend. In acute hemorrhagic and embolic defibrination of abruptio placenta, efforts to interrupt the pregnancy other than amniotomy should be delayed until adequate supportive treatment has been given. Fresh whole blood and 4 to 10 Gm. of fibrinogen may be necessary for emergency treatment.

For many years obstetricians have avoided surgical interference when there is a dead fetus in the uterus. Ninety per cent of instances in which the fetus dies in utero will be followed by labor within 4 weeks without the risk of maternal blood incoagulability. Surgical induction of labor is attended by certain risks of infection, particularly from gas gangrene organisms. A hysterotomy seems unnecessarily radical. It is probably wise to determine the fibrinogen level weekly and follow a policy of noninterference as long as the plasma fibrinogen level ranges above 150 mg. of fibrinogen per 100 cc. of plasma. If the fibrinogen level drops to between 150 and 90 mg., the frequency of blood sampling should be increased to several times daily and preparation made for delivery. The method of delivery depends on obstetric conditions. A long hard cervix, in the judgment of some physicians, calls for hysterotomy or even hysterectomy. A cervix favorable for delivery would indicate amniotomy followed by Pitocin drip. In any case, amniotomy is wise regardless of the condition of the cervix to discourage further absorption of amniotic fluid containing material rich in thromboplastin. The prophylactic use of fibrinogen is discouraged because of the risk of homologous serum jaundice. It also must be remembered that fibrinogen will give only temporary elevations of the fibrinogen level unless the pregnancy is interrupted.

In general, the following rules should be observed in all cases of hemorrhage not promptly controlled or in which a coagulation defect might reasonably be suspected:

1. Ten cc. of blood should be withdrawn into an oxalated tube and submitted to a laboratory for testing. Two cc. of blood should be placed in two scrupulously clean serology tubes. The blood should be timed from the moment the blood appears in the syringe. The first tube should be gently tilted every 5 minutes until clotting occurs. Then the second tube should be checked in the same manner. The coagulation time is measured from the appearance of blood in the syringe to the clotting of the blood in the tube.

2. Coagulation time of 15 to 20 minutes is borderline. Anything more than 20 minutes is pathological. The tube then should be observed for dissolution of the clot. If the clot in both tubes dissolves within 30 minutes, the blood fibrinogen level is approximately 75 to 100 mg. per 100 cc. Adequate and effective treatment will require from 1 to 4 mg. of fibrinogen. In addition, of course, whole fresh blood should be given to the patient for blood replacement.

SPECULATION

The physiological and pathological mechanism of fibrinogen-fibrin reaction leads to many interesting speculations. Nisely's work on sludged blood suggests that fibrin plays an important role in the sludging phenomena. Pituitary necrosis, bilateral renal cortical necrosis, and certain forms of cerebral thrombosis may well be related to organ insufficiency from fibrin embolization. Convulsive eclampsia and unusually severe uterine contraction may serve as a defibrinating factor. The role of exertional muscular trauma in the nonconditioned musculature of a desk executive may well play a role in the vacation coronary or cerebral thrombosis. It is also interesting to speculate on the defibrinating role of surgical trauma, which may serve as a nidus for subsequent phlebothrombosis and pulmonary embolism.

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Clinical Evaluation of Su-5879 (Esidrix), A New Oral Sulfonamide Diuretic

Preliminary Report

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THE EFFECTIVENESS of the sulfonamide derivatives in the diuretic therapy of edematous states has been well documented. The synthesis of acetazolamide, which acts as a carbonic anhydrase inhibitor,[1, 5] was the first dramatic step in the clinical utilization of this group of compounds. The next development was the introduction of chlorothiazide,[2, 3] which is of low toxicity and acts in a manner similar to the mercurial diuretics. Furthermore, chlorothiazide has been shown to be of considerable value in the therapy of hypertension.[6] The most recent advance has been the development of dihydrochlorothiazide.[4]

Such developments have stimulated the search for new and possibly more effective diuretic and antihypertensive drugs in the sulfonamide group. Su-5879, or Esidrix, Ciba's dihydro-chlorothiazide, is such a compound, and is chemically closely related to chlorothiazide (fig. 1). Preliminary dog experiments[4] showed Su-5879 to be a very potent diuretic substance, of unusually low toxicity. Although only one-ninth as potent as chlorothiazide in capacity to inhibit carbonic anhydrase, it seemed to be from 7 to 16 times as effective in achieving diuresis in the dog and rat, and to have a longer duration of diuretic action. The animal experimental studies also appeared to show less potassium dissipation than chlorothiazide in equipotent dosage.

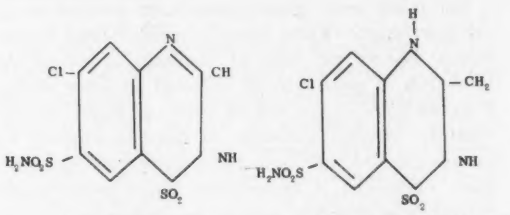

Fig. 1. Formulas showing the chemical relationship of chlorothiazide and dihydro-chlorothiazide (Su-5879, Esidrex).

Dr. George R. Herrmann and his co-authors presented this report from the Cardiovascular Service of the University of Texas Medical Branch at a meeting of the Texas Academy of Internal Medicine, December 6, 1958, in Galveston.

Su-5879 (Esidrix) or dihydro-chlorothiazide, a new sulfonamide, proved an effective oral diuretic of low toxicity for 21 patients with edema. Necessary dosage was one-fourth to one-eighth that of chlorothiazide. Excretion of urine and electrolytes was similar to the effect of chlorothiazide. Antihypertensive action appears promising, but nitrogen retention calls for care in long term therapy.

METHOD OF STUDY

We have determined the effects of Su-5879 on the electrolyte patterns in the urine and plasma during clinical diuresis in 14 hospitalized and in 7 outclinic patients with edema. Each of our studies on inpatients was begun with a control period of 3 to 4 days, during which the regimen of therapy for the congestive heart failure was kept constant, a low (500 mg.) sodium diet was administered, and all other diuretic agents and potassium were withheld. During this period there was careful clinical evaluation, with determination of daily body weight, complete blood count, urinalysis, venous pressure, blood urea nitrogen, and serum sodium, potassium, carbon dioxide, and chloride. Urine specimens were collected and measured, and the content of sodium, potassium, and chloride determined for the 12 hour period, and for a 2 hour period immediatedy prior to the oral administration of Su-5879.

The diuretic then was given in initial dosage of 300 mg. in 7 patients and later reduced to 200 mg., which was equally effective in 7 patients. Urines were collected over 2-hour periods for the next 10 hours, measured, and analyzed for content of sodium, potassium, and chloride. Then a 12-hour specimen and thereafter 24-hour specimens were collected and

analyzed similarly. After 48 hours, the patients were placed on a maintenance regimen of 50 mg. of Su-5879, 3 times daily in 7 patients who had received the higher initial dose and 2 times daily in the other 7 patients. The progress of each hospital patient was clinically followed, and the body weights were determined daily. Serum electrolyte studies and blood urea nitrogen determinations were made at the end of the initial 48 hour period of study and weekly thereafter. Follow-up blood counts, urinalyses, and venous pressures were determined weekly.

The outpatients were in less severe failure and were evaluated 1 to 2 times weekly, but urinary volume and urinary electrolyte determinations were not done. In these patients, Su-5879 was given in doses of 50 mg. twice daily. Complete blood count, urinalysis, blood urinary nitrogen, and serum sodium, potassium, carbon dioxide, and chloride determinations were done prior to administering the diuretic, at each clinic visit thereafter for the first several weeks, and periodically later.

CLINICAL RESULTS

There were 20 patients in congestive heart failure with arteriosclerotic coronary heart disease in 12, hypertensive in 3, rheumatic in 3, syphilitic in 1, and heart disease of unknown cause in 1. One patient had edema of undetermined origin. The venous pressures in the 20 patients in congestive failure ranged from 15 to 31 cm. of saline, the average being 23.4 cm. Blood urea nitrogen values initially varied from 13 to 44 mg. per 100 cc. Blood counts were within normal limits, except in 1 patient with myelofibrosis. Serum protein values were also normal in all cases.

Satisfactory clinical responses were obtained in all except 1 hospitalized patient (the 1 with myelofibrosis), who did not increase the secretion of urine. In all others, there was clearing of the symptoms and signs of congestive heart failure in a period of from 1 to 10 days. Venous pressures were returned to within normal limits (less than 15 cm. saline) in all patients except in the patient who did not increase the output of urine.

There were few side effects. Two patients (1

TABLE 1.—*Urine and Electrolyte Excretion After Administration of Su-5879.*

	Before Su-5879		After Beginning Su-5879										
	12 hr. Control	2 hr. Control	0-2 hr.	2-4 hr.	4-6 hr.	6-8 hr.	8-10 hr.	10-22 hr.	2nd Day	3rd Day	4th Day	5th Day	6th Day	7th Day
Urine (cc./hr.)	58	69	69	115	97	101	88	118	122	98	111	114	89	89
Na (meq/hr.)	3.2	3.5	5.1	11.3	11.3	11.6	9.5	10.6	7.7	9.2	8.0	6.8	6.4	4.3
K (meq/hr.)	2.3	2.3	2.1	3.0	2.1	3.1	2.6	2.5	2.1	2.6	2.7	3.1	3.0	2.8
Cl (meq/hr.)	3.5	4.3	5.4	11.2	11.6	12.0	10.4	12.7	7.5	9.5	8.2	6.4	5.8	5.6

TABLE 2.—*Serum Electrolyte and Carbon Dioxide Levels Before and After Administration of Su-5879.*

	Before				After				
Case	Na (meq/L)	K (meq/L)	Cl (meq/L)	CO_2 (meq/L)	Na (meq/L)	K (meq/L)	Cl (meq/L)	CO_2 (meq/L)	Days of Therapy
				HOSPITAL PATIENTS					
1	130	6.1	93.5	19.7	137	4.1	99	19.7	4
2	127	4.7	100	26	147	5.0	99	17.5	18
3	143	4.4	102	25.2	135	5.7	90.5	21	4
4	143	5.4	108	21.7	147	5.5	104	27	4
5	141	4.4	112	21.0	143	6.0	108	34	2
6	139	6.2	109	25.2	150	5.0	106	28	5
7	150	5.1	107	21.0	140	4.2	97	28.2	17
8	142	4.6	113	17.5	140	4.0	101	26.8	6
9	147	4.3	101	18.6	146	5.3	108	31.1	2
10	141	4.7	108	24.4	144	5.4	102	30.7	2
11	149	4.6	108	27.3	140	4.3	99.1	31.1	4
12	140	5.3	111	30.2	146	6.9	92	32.4	5
13	150	5.3	105	32.4	143	4.8	96	25.6	4
14	134	4.2	104	21.0	145	4.9	106	21.8	2
				CLINIC PATIENTS					
15	152	4.9	109	26.0	143	4.5	98	32.8	20
16	146	6.1	113	26.5	146	5.1	104	36.1	7
17	147	4.1	100	21	148	4.6	108	29	14
18	146	3.5	109	28	152	4.6	96	36.1	14
19	144	4.3	101	28.7	146	4.3	98	30.2	14
20	140	3.8	108	28.2	143	5.0	95	30	7
21	152	4.9	109	26.0	143	4.5	98	32.8	21

hospitalized and 1 outpatient) had nausea and vomiting after taking the drug. In the hospitalized patient, these symptoms when present seemed definitely to be due to the congestive heart failure state. In the outpatient, Su-5879 apparently was not the cause since the medication was later resumed without symptoms. In most cases, elevated blood urea nitrogen levels returned to normal as compensation was established, except in 2 hypertensive patients whose initial blood urea nitrogen levels were normal, in whom there was a moderate increase in blood urea nitrogen which persisted. Urines nearly always were acid in reaction after the drug was given, and there were no adverse urinary changes.

Diuretic action in the hospitalized patients resulted in an average weight loss in the first 24 hours of 3.1 pounds (varying from 0 in the nonresponsive patient to a maximum of 8 pounds). The average weight and edema loss in the first week amounted to 13.1 pounds (varying from 0 in 1 patient to a maximum of 33 pounds). All of the outclinic patients increased their urine secretion, with an average of 4.4 pounds weight loss in the first week and with circulatory equilibrium reestablished in all by the end of the first week of therapy.

Table 1 shows the average urine volume, sodium excretion, potassium excretion, and chloride excretion in the 14 hospitalized patients before and after the administration of Su-5879.

This table shows the urinary volume to increase in the second 2-hour period following the administration of a single dose of 300 or of 200 mg. of Su-5879 and to continue at increased amounts for 48 hours despite the fact that no more drug was administered. Increased amounts of urinary volume were also recorded from the third to seventh day, during which time the patients were receiving 50 mg. of Su-5879 2 to 3 times daily.

The excretion of sodium increased by about 45 per cent in the first 2 hours, and then tripled in the following 2-hour and 24-hour periods, remaining well over the initial levels throughout the week. Potassium excretion increased to a maximum of 30 per cent in the 2 to 4 and 6 to 8 hour periods, but was

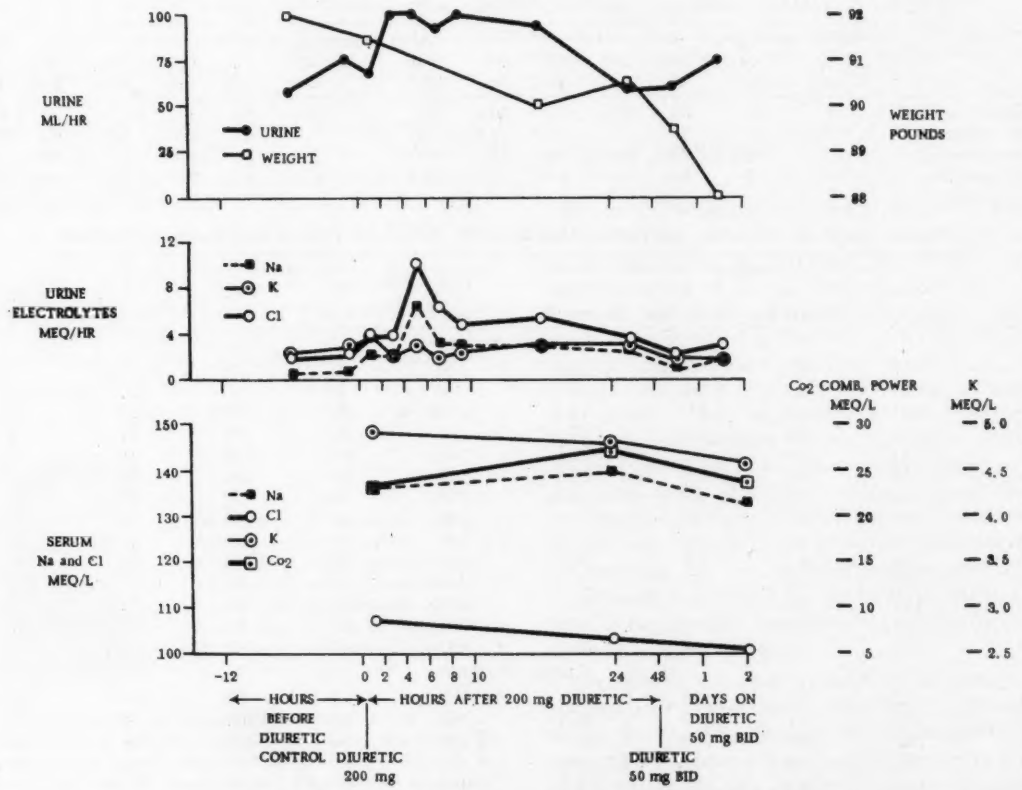

Fig. 2. Diuretic study in a 24 year old woman with rheumatic heart disease and failure to whom Su-5879 was administered.

generally below the sodium level. The chloride output was augmented to essentially the same degree as the sodium.

The serum electrolyte and carbon dioxide levels before and after the administration of Su-5879 are summarized in table 2. Serum sodium levels were within normal limits after treatment and actually were increased in the 2 cases in which the initial values were low. Serum potassium levels decreased in 9 patients, in 3 of whom the drug had been administered more than 2 weeks. Four of these patients showed serum potassium levels less than 4.5 milliequivalents per liter, but none less than 4.0 milliequivalents per liter. Serum chloride levels decreased in all but 4 patients, to below 95 milliequivalents per liter in 2 patients, but in none to less than 90 milliequivalents per liter. Serum carbon dioxide levels increased in 17 patients, to more than 30 milliequivalents per liter in 10, and to more than 35 milliequivalents per liter in 2 cases.

Figure 2 shows the weight, urinary volume, electrolyte excretion, and serum carbon dioxide and electrolyte levels before and after the administration of Su-5879 to a patient who demonstrated an effective clinical response to the drug.

ANTIHYPERTENSIVE ACTION

Blood pressures were carefully determined as part of the clinical evaluation in all patients receiving Su-5879. Of the 3 hypertensive patients in the series, a hospitalized patient showed a fall of blood pressure from 170/110 mm. of mercury to 130/80 on Su-5879 alone. Another patient in heart failure with essential hypertension seen in the Outpatient Clinic showed a fall of 40/30 from 190/110 to 150/80 when Su-5879 was added to his previous Su-3118 (Ciba, Singoserp) 1 mg. three times daily, to which he had not responded. An outpatient with malignant hypertension showed no decrease from levels of 290/130 on Su-3118 1 mg. four times daily and Su-5879 three times daily. Two outpatients showed lowerings of blood pressures from 174/130 to 130/90 on Su-5879 alone given three times daily, and from 186/124 to 170/110 when Su-5879 was added to the previous regimen of Su-3118 1 mg. three times daily. Another outpatient showed a change from 190/100 to 150/100 when given Su-5879 three times daily without other medication.

DISCUSSION

Su-5879 appears to be a clinically effective oral diuretic which is well tolerated. The initial oral doses of 300 mg. for hospitalized patients and maintenance doses of 50 mg. three times daily were apparently higher than necessary in most cases, and the later regimen of 200 mg. for initial action and 50 mg. twice daily was just as effective in our cases.

The diuretic effect appears to be similar to that of the mercurials, in that urinary volume is augmented, with predominantly an excretion of sodium and chloride to about three times the control levels. Potassium excretion was increased slightly to a maximum increase of 30 per cent in these short term studies.

The reaction of the urine was nearly always acid, and blood electrolyte determinations usually showed a decrease in chloride and an increase in carbon dioxide values. Serum potassium was decreased in some patients, but clinically significant or symptomatic hypopotassemia was not observed in any of these patients intensively treated, usually for 2 weeks, to dissipate the edema. Five of our patients have now been maintained on the drug for as long as a month with lowered serum levels but without any serious potassium depletion.

Blood urea nitrogen levels rose moderately and persisted in 2 patients, and more intense and longer term studies are necessary to determine the incidence and significance of such rises. It is possible that latent renal failure was uncovered by diuresis. Blood pressure follow-ups offer considerable promise that Su-5879 will be an effective antihypertensive drug, as the pressure was lowered in all patients except 1 with malignant hypertension and to normal levels in 3 of 7 patients. Hypertensive patients with slight renal failure may suffer from increased irritation and nitrogen retention just as such patients do on chlorothiazide therapy.

SUMMARY

Su-5879 (Esidrix), or dihydro-chlorothiazide, a new sulfonamide compound, has been demonstrated to be an effective, oral diuretic of very low toxicity in studies on 21 patients with edema. The dosage necessary was one-fourth to one-eighth that of chlorothiazide, and so milligram for milligram, Su-5879 is the most effective oral diuretic that we have used.

The diuretic effect is similar to that of the mercurials and chlorothiazide, in that there results mainly an outpouring of sodium and chloride, with decrease of chloride levels and increase of carbon dioxide in the serum.

A single dose of 200 mg. produces a diuresis which begins within about 2 hours after one ingestion and is maintained for more than 24 hours. Effective diuresis also can be achieved by 50 mg. doses administered twice daily.

Potassium excretion is increased, almost as much

as it is with chlorothiazide, but no significant hypopotassemia was observed in these short term studies.

On the basis of a limited number of cases, the antihypertensive action of Su-5879 appears promising. However, the matter of nitrogen retention calls for care in long term therapy in hypertensive patients, especially those with renal diseases, just as is the case with chlorothiazide.

Oliguria and rising blood urea nitrogen levels call for withdrawal of the drug.

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Correction: Cervix Carcinoma

Figure 3 illustrating the article on "Mismanagement of Carcinoma of the Cervix" by Drs. Felix N. Rutledge and James Roderick Morgan in the November issue of the *Journal*, pages 769-773, was printed upside down. The drawing, on page 772, should have appeared as follows:

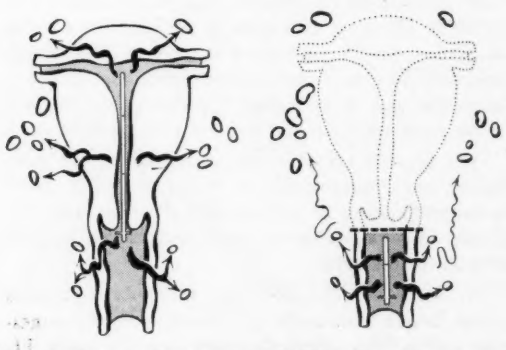

Fig. 3. Prior to hysterectomy **(left)** radium therapy can reach regional lymph nodes to combat metastasis with little difficulty, but after hysterectomy **(right)** the problem is more difficult.

CASE REPORTS

Acute Hemorrhagic Pericarditis with Cardiac Tamponade

Report of a Case with Recovery After Pericardicentesis

JAMES E. KREISLE, M.D., and MATTHEW F. KREISLE, SR., M.D.

Austin, Texas

THERE HAVE RECENTLY been reported 4 instances of fatal cardiac tamponade occurring in the course of acute fibrinous pericarditis of undetermined etiology.[7, 11, 14, 16] In all 4 of these cases the pericardial fluid was grossly bloody.

The patient described hereafter developed severe hemorrhagic tamponade in the course of an illness, the clinical features of which appeared consistent with "acute nonspecific pericarditis." In view of this patient's prompt recovery following pericardicentesis, it is thought that a detailed account of the case may be of interest.

CASE REPORT

J. B. M., a 60 year old male mental hospital attendant, was well until January 25, 1957, when, while walking rapidly out of doors, he began to notice with each breath a sharp pain in the region of the suprasternal notch. Over the next few days the pain became more severe, spread to involve the whole sternal area, and finally extended over the right lower pectoral region into the axilla. Even shallow inspiration accentuated the pain. He continued work, however, until January 31, when we were called to see him at his home about 2 a.m. He was in moderately severe respiratory distress because of "pleuritic" type pain substernally and in the right lower pectoral and axillary regions. The pain was aggravated in the recumbent position. There was a slight hacking cough. The temperature was 101.5 F. Breath sounds were markedly suppressed over the right lower portion of the chest anteriorly, and a few crepitant rales were heard in the same area. No pleural or

pericardial friction rub was detected. An electrocardiogram taken later in the morning is shown in figure 1a.

The patient refused to be hospitalized and so remained in bed at home. Tetracycline, 250 mg. every 4 hours, and analgesic medication were prescribed. At this point contact with the patient was inadvertently lost, as he failed to report his condition as instructed. During the next few days his pain subsided, and he gained the impression that his fever was abating. Hence, on February 7, he returned to work despite the fact that his evening temperature was 100.6 F. and he was experiencing chilly sensations and night sweats.

Back at work he found himself quite short of breath on effort and noticed that he gradually gained 10 pounds even though he ate very little. He developed a full feeling in the upper part of the abdomen, more intense on the right side. Slight cough persisted, but chest pain did not recur. Orthopnea was not noted. Sometimes there was considerable dyspnea after fits of coughing.

On February 25 the patient had to leave work because of complete exhaustion. Also, he had had several episodes of sudden pressure about the lower ribs, along with a sense of inability to breathe; on suddenly getting his breath he would feel as if blood rushed to his head and would notice blurring of vision for a few seconds. When seen by us at home he was in apparent congestive failure and so was hospitalized at once.

Family and marital histories revealed no relevant data. The patient had been employed at several hospitals during his life but did not know of any contact with tuberculous patients. He had had the usual childhood illnesses but gave no history of rheumatic fever. He had had pneumonia in 1924. No history of hypertension, coronary disease, or heart murmur was elicited. For more than 20 years prior to his present illness he had not been sick.

Physical examination at the time of hospital admission on February 25 showed a large, rather heavy-set elderly man coughing occasionally and breathing in slightly labored fashion. The blood pressure was 120/90 mm. of mercury, and the pulse was regular at 100 per minute. There was marked diminution of pulse volume during inspiration (paradoxical pulse). The skin was slightly pale but neither cyanotic nor clammy. The neck veins were full, even when the patient sat upright. Cardiac percussion dullness extended to the midclavicular line on the right and to the anterior axillary line on the left. No point of maximum cardiac impulse could be determined, but a weak, diffuse systolic impulse was felt over the precordium. The heart sounds were faint, being most distinctly heard in the left anterior axillary region. No murmur, friction rub, or gallop rhythm was noted. Coarse, crepitant rales were heard at the right lung base; there was no impairment of resonance, and breath sounds were of normal quality and intensity throughout. The liver edge was moderately tender and extended 3 to 4 fingerbreadths below the costal margin. There was no dependent edema or calf tenderness. The remainder of the examination was negative.

Dr. James E. Kreisle and his coauthor, Dr. Matthew F. Kreisle, Sr., specialize in the practice of internal medicine.

A case of acute hemorrhagic pericarditis with tamponade in a 60 year old man with recovery following pericardicentesis is reported. Possible identity of this disease with acute nonspecific or benign pericarditis is considered. Reports of fatal cardiac tamponade in acute nonspecific pericarditis are reviewed. The significance of marked elevation of serum transaminase activity is discussed.

Initial laboratory data were as follows: red blood cells, 3,730,000 per cubic millimeter; hemoglobin, 9.3 Gm. per 100 cc.; white blood cells 11,200 per cubic millimeter with 66 per cent segmented neutrophils, 4 per cent stabs, 25 per cent lymphocytes, and 5 per cent monocytes; sedimentation rate (Wintrobe), 40 mm. per hour; blood platelets, 301,000 per cubic millimeter; bleeding time, 2 minutes; clotting time, 4 minutes; urinalysis, specific gravity 1.027, pH 5.0, very faint trace of albumin, sugar negative, acetone negative, 1 hyaline cast, and 1 or 2 white blood cells per high power field in urinary sediment; blood urea nitrogen, 25.6 mg. per 100 cc.; creatinine, 1.2 mg. per 100 cc.; carbon dioxide combining power, 18.5 milliequivalents per liter; chloride, 102 milliequivalents per liter; prothrombine time, 17 seconds (control, 13 seconds); serum transaminase, 140 units; serum VDRL and Mazzini tests, nonreactive.

An electrocardiogram recorded on the day of admission is shown in figure 1b.

Roentgen-ray examination of the chest, including fluoroscopy, showed marked enlargement of the cardiac shadow in all diameters. There was normal amplitude of pulsation along the left heart border but marked reduction in pulsation of the right border. The lung fields were clear. No pleural fluid was observed. (See figure 2.)

Initial therapy consisted of bed rest with head elevation, oxygen by nasal cannula, low sodium diet, digitalization, and 2 cc. of Mercuhydrin intramuscularly.

During his first 4 days in the hospital the patient ran slight afternoon fever ranging from 101 F. on the day of admission down to 99.2 F. on the fourth day; the remainder of his hospital course was afebrile. There was little change in his condition until 7 a.m. on February 27, at which time he was found to be lethargic and to have a rapid, weak, somewhat irregular radial pulse. A nurse recorded the blood pressure as 100/60 mm. of mercury. The respiratory rate, previously around 24 per minute, had increased to 40 per minute. By 8:30 a.m. we found his radial pulse almost imperceptible. The neck veins were full, the heart sounds were extremely faint, and no change in lung signs was noted. An electrocardiogram recorded at this time is shown in figure 1c. Three attempts at pericardial tap were made in the fourth intercostal space just medial to the left border of dullness. The first two attempts yielded small amounts of dark blood, but on the third trial a free flow of what appeared to be venous blood was obtained. After the aspiration of about 50 cc. the needle was withdrawn, as it was feared the right ventricle had been entered. Almost immediately, however, the patient began to improve. Just after the tap his blood pressure was 120/80 mm. of mercury, and his pulse became stronger and slower. At 9 a.m. his blood pressure was 110/70 mm. of mercury, pulse rate 70 per minute, and respiratory rate 28 per minute. At 7 p.m. on the same day an additional 130 cc. of dark, thick, bloody fluid was aspirated from the pericardial space.

The leukocyte count on the day of pericardial tap was 14,400 per cubic millimeter with 78 per cent neutrophils, 14 per cent lymphocytes, 6 per cent monocytes, and 2 per cent eosinophils. Following the procedure the patient was

given tetracycline, 100 mg. intramuscularly every 6 hours for 1 week.

On February 28, the day after aspiration, the patient was remarkably better. His neck veins were collapsed, and his pulse was no longer paradoxical. Cough and dyspnea on effort were his only symptoms. From this point on he continued to show steady and rapid improvement. The extent of cardiac dullness decreased, the heart sounds became louder, the blood pressure rose to 140/80 mm. of mercury, and the liver became smaller and less tender. Urinary output was well in excess of fluid intake for a week. On March 1 signs of left pleural fluid, confirmed by roentgen ray (fig. 3 left), were noted. On only one day, March 3, was a distinct pericardial friction rub detected. Chest roentgenograms on March 8 showed marked reduction in the size of the cardiac shadow and diminution in the amount of fluid in the left pleural space (fig. 3 right). Electrocardiograms recorded during the period of convalescence are shown in figures 1d, e, and f. Cough and dyspnea were gone by March 10. Slight hepatic enlargement was still present at the time the patient was discharged on March 17, his twenty-first hospital day. By the time of discharge the red blood count was 4,050,000 per cubic millimeter, hemoglobin 11.4 Gm. per 100 cc., sedimentation rate (Wintrobe) 32 mm. per hour, and white blood count 8,000 per cubic millimeter with a normal differential count.

Of special interest was the serum transaminase level (Leeper method), which showed the following variations: February 25, 140 units; February 26, 300 units; March 1, 370 units; March 4, 190 units; March 13, 25 units. It was believed that these values probably reflected hepatocellular damage due to venous congestion. Other evidences of liver impairment were as follows (March 6): total bilirubin, 1.1 mg. per 100 cc. (direct 0.3 mg. and indirect 0.8 mg.); thymol turbidity, 10 units; cephalin flocculation, 3 plus at

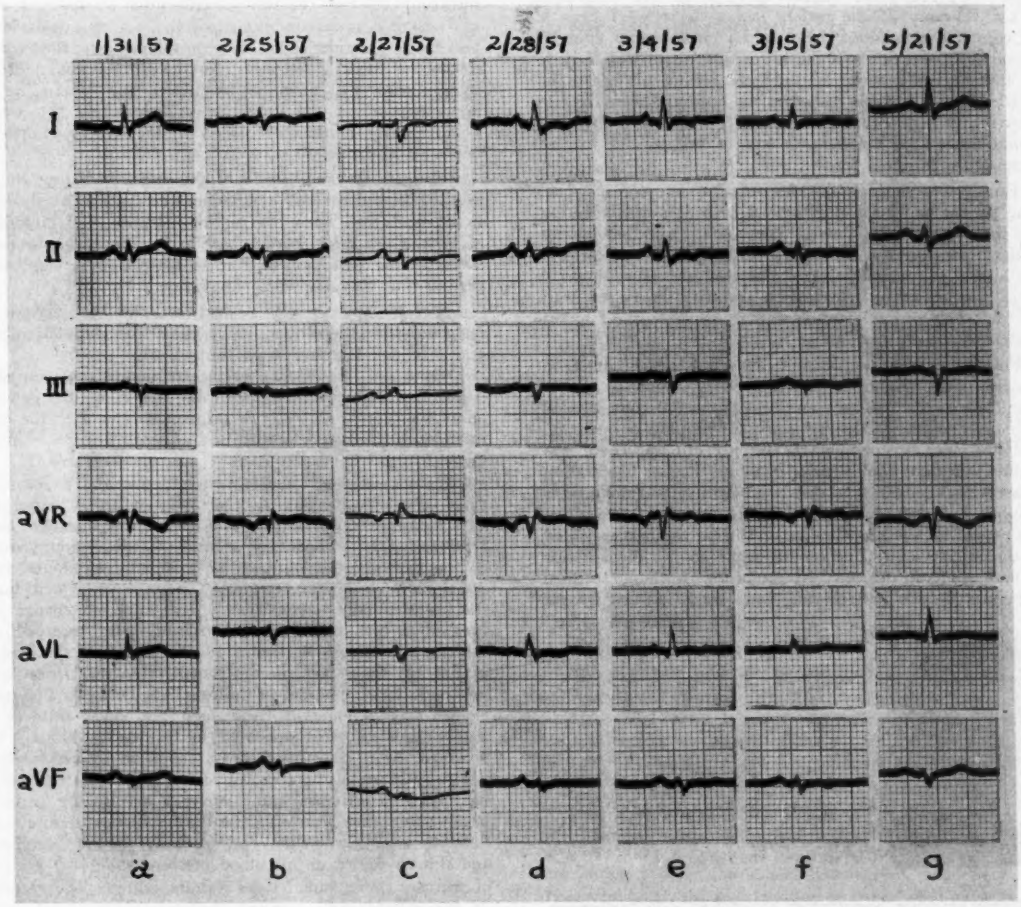

Fig. 1. Serial electrocardiograms in acute hemorrhagic pericarditis with cardiac tamponade: **(a)** tracing made 6 days after onset of symptoms, showing slight elevation of ST segments as compared with final tracing shown in **g**; **(b)** recorded on hospital admission 31 days after onset of symptoms; **(c)** recorded during period of critical tamponade just prior to pericardicentesis; **(d)** recorded on day following pericardicentesis; **(e, f)** recorded during period of convalescence in hospital; **(g)** recorded 4 months after onset, when apparent complete recovery had occurred.

24 and 48 hours; prothrombin time, 15 seconds (control 12 seconds); total serum protein, 7.6 Gm. per 100 cc. with 3.6 Gm. of albumin and 4.0 Gm. of globulin.

Following discharge from the hospital the patient had an uneventful convalescence. When he was examined on May 21, 4 months after the onset of his illness, he was back working full time and seemed in excellent health. The blood pressure was 142/90 mm. of mercury, the pulse rate 84 per minute, and the afternoon temperature 98.6 F. The heart was not enlarged to physical examination, the heart sounds were of good intensity, the aortic and pulmonic second sounds were equal, and no murmurs were heard. The lungs were clear except for a few coarse crepitant rales at the left base. An electrocardiogram recorded at this time is shown in figure 1g.

The patient did not return for further follow-ups. However, his employer confirmed the fact that he worked and was apparently well until October 6, 1957, at which time he moved to another community.

COMMENT

In the strict sense the reported case must be considered an instance of intrapericardial hemorrhage of uncertain cause. The possibility of dissecting aortic aneurysm with hemopericardium was considered;

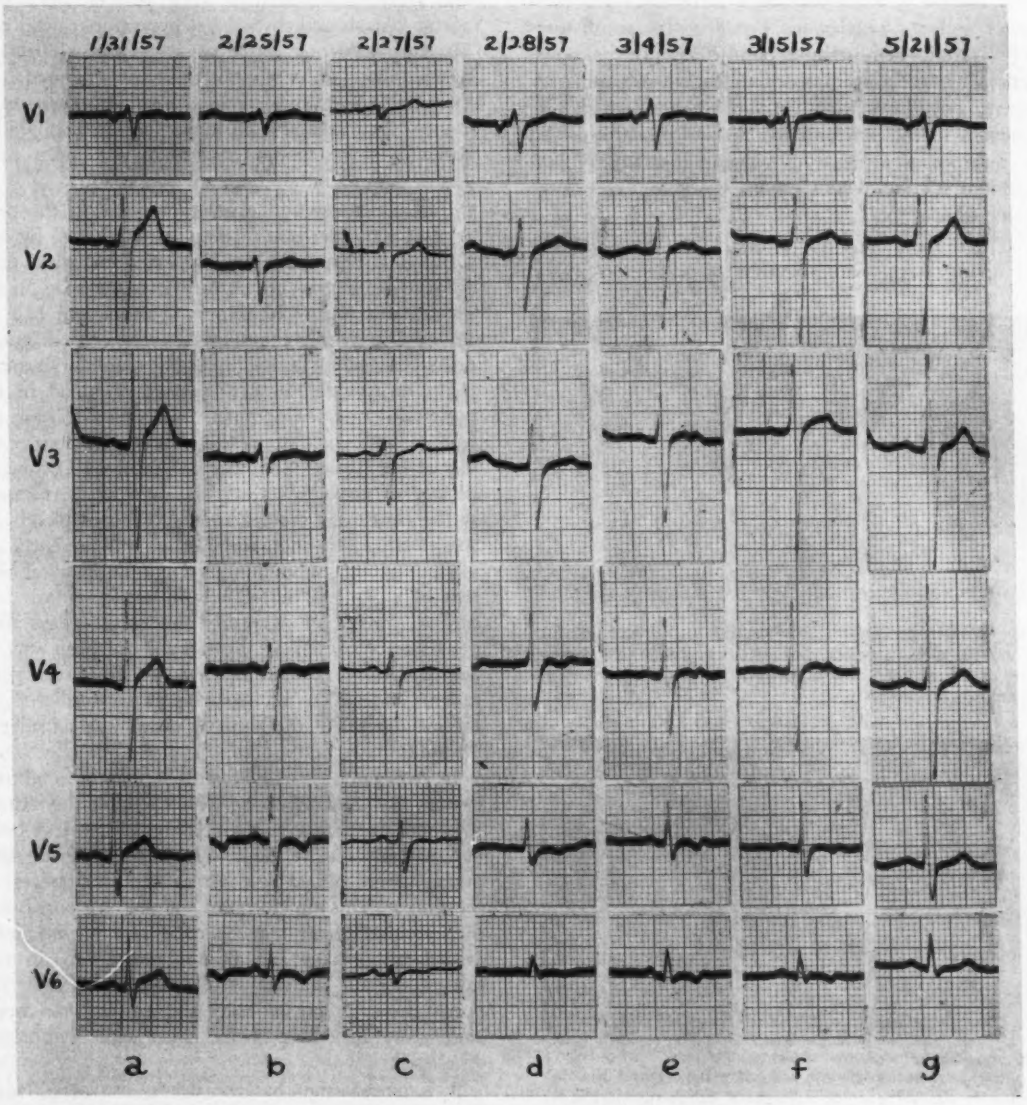

Fig. 1. (continued). Serial electrocardiograms in acute hemorrhagic pericarditis with cardiac tamponade.

however, only a single patient with this condition is known to have survived for more than 5 weeks.[9] Likewise, the prompt recovery of our patient following relief of tamponade renders unlikely such serious conditions as tuberculosis and malignant neoplasm. We believe that the clinical picture is perhaps most consistent with what is referred to in the literature as acute "nonspecific," "idiopathic," or "benign" pericarditis.[2, 5, 8, 17]

There is ample evidence for the existence of a hemorrhagic phase in acute nonspecific pericarditis. Pericardial fluid obtained after the first 10 days of the disease is commonly of a serosanguinous character.[12, 13, 15, 17] Effler and Proudfit observed 8 cases of "chronic hemorrhagic pericarditis" of unknown cause;[6] in their opinion some obscure inflammatory agent, possibly a virus, had provoked hemorrhagic changes in the pericardium. Yu and others reported a remarkable instance of chronic massive serosanguinous effusion with signs and symptoms of cardiac compression.[18]

There are 2 reports of fatal cardiac tamponade occurring in the natural course of acute nonspecific pericarditis. Pomerance and associates[14] described the case of a 46 year old woman who died only 6 days after the onset of symptoms; the clinical impression was acute pancreatitis. At autopsy the pericardial sac contained 500 cc. of serosanguinous fluid, and the pericardium showed fresh fibrinous exudate superimposed upon a chronic inflammatory process. Price and others[16] reported the case of a 22 year old man who died in acute renal failure on about the thirty-fifth day of his disease despite 2 pericardial taps yielding a total of 960 cc. of serosanguinous fluid. At autopsy the pericardial cavity contained 1,000 cc. of blood-stained fluid, and lower nephron nephrosis was present. Case 44201[4] of the Massachusetts General Hospital was that of a 64 year old man with aortic stenosis and myocardial hypertrophy who died 30 days following onset of acute pericarditis. Although 40 cc. of sanguinous fluid was aspirated from the pericardium ante mortem, and autopsy revealed "acute hemorrhagic fibrinous pericarditis (idiopathic)," it was thought that death was due to myocardial failure rather than to tamponade.

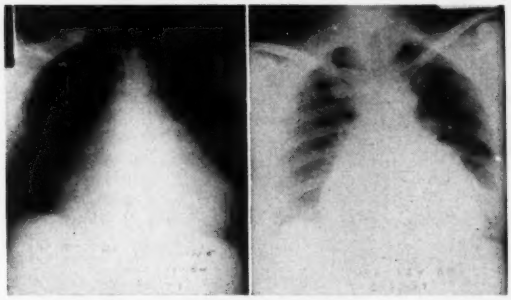

Fig. 2. Roentgenograms showing enlargement of cardiac silhouette due to pericardial effusion, made on admission of the patient to the hospital: left, supine position; right, erect position.

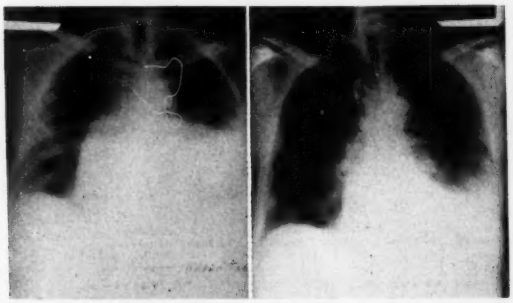

Fig. 3. Roentgenograms showing the development and beginning resolution of left pleural effusion and the decrease in size of cardiac silhouette following pericardicentesis.

The danger of anticoagulant therapy in the presence of acute pericarditis was first suggested by McCord and Taguchi,[11] who reported the case of a 52 year old man dying of cardiac tamponade on the seventeenth day of his illness. This patient had received Dicumarol because of an erroneous diagnosis of myocardial infarction. Autopsy revealed fibrinous pericarditis with 150 cc. or more of blood in the pericardial cavity. Goodman[7] observed a 76 year old woman who died of tamponade on the fourteenth day of her disease; she, too, had received Dicumarol because of suspected myocardial infarction. At autopsy the pericardial sac contained 700 cc. of blood and soft clots, and fibrinous pericarditis was present. Case 42151[3] of the Massachusetts General Hospital was that of a 70 year old man with aortic stenosis and cardiac hypertrophy who died suddenly 14 days after the onset of chest pain. Dicumarol had been administered. Although autopsy showed fibrinous pericarditis and 600 cc. of hemorrhagic pericardial fluid, the mechanism of the patient's death was considered uncertain.

In our case, although no anticoagulant was administered, critical tamponade developed on the thirty-third day of illness. It may be significant that both our patient and the one described by Price and others[16] were not at rest during the early stages of the disease. This circumstance suggests the possibility that physical effort may be conducive to intrapericardial hemorrhage and fluid accumulation. In the case of chronic massive serosanguinous effusion reported by Yu and others,[18] the patient had been ambulatory for more than a year after symptoms developed.

It is noteworthy that the deaths associated with the use of Dicumarol occurred 14 to 17 days after

the onset of symptoms,[7, 11] whereas 2 of the patients (ours and that of Price and others[16]) with spontaneous tamponade did not progress to a critical state until after the thirtieth day of illness. The case of spontaneous tamponade reported by Pomerance and others[14] was stated to have been symptomatic for only 6 days prior to death, but the condition of the pericardium at autopsy clearly indicated a disease of much longer duration.

Our patient improved steadily after the aspiration of a relatively small volume of pericardial fluid. The prompt appearance of left pleural effusion following pericardicentesis suggests that drainage from the pericardial space into the left pleural cavity developed fortuitously after the taps, thus allowing a more effective decompression to occur. Effler and Proudfit have shown the efficacy of such a mechanism in the surgical drainage of chronic pericardial effusions.[6]

Slight to moderate increase in serum glutamic oxalacetic transaminase activity is reported to occur in both experimental[1] and clinical[10] pericarditis; this phenomenon has been attributed to subepicardial myocarditis. The maximum level noted by Kalmansohn and Kalmansohn in a case of nonspecific pericarditis was 165 units (Karmen method). In our patient the maximum level was 370 units (Leeper method), and the elevation was much more sustained than is characteristic of myocardial infarction; our impression is that the increase in transaminase activity roughly paralleled the degree of hepatic congestion.

SUMMARY

A case is presented in which recovery from acute hemorrhagic pericarditis with tamponade occurred following pericardicentesis.

The possible identity of the patient's disease with acute nonspecific or benign pericarditis is considered.

The reported cases of fatal cardiac tamponade in acute nonspecific pericarditis are reviewed.

Marked elevation of serum transaminase activity in the presented case is reported, and the significance of this finding is discussed.

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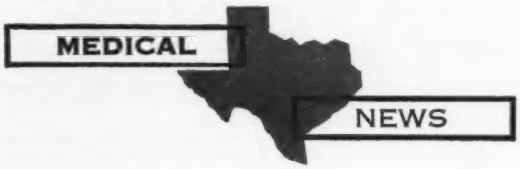

★ Coming Meetings

Texas Medical Association, San Antonio, April 18-21, 1959 (Conference of County Medical Society Officials and Symposium on Legislation, Jan. 24, 1959). Dr. Howard O. Smith, Pres.; Mr. C. Lincoln Williston, 1801 North Lamar Blvd., Austin, Exec. Secy.

American Medical Association, Clinical Meeting, Minneapolis, Dec. 2-5, 1958. Dr. Gunnar Gundersen, La Crosse, Wis., Pres.; Dr. F. J. L. Blasingame, 535 North Dearborn, Chicago 10, Exec. Vice-Pres.

Current Meetings

DECEMBER

American Academy of Dermatology and Syphilology, Chicago, Dec. 6-11, 1958. Dr. Everett R. Seale, Houston, Pres.; Dr. R. R. Kierland, Mayo Clinic, Rochester, Minn., Secy.

Southern Surgical Association, Boca Raton, Fla., Dec. 9-11, 1958. Dr. James D. Rives, New Orleans, Pres.; Dr. George G. Finney, 2947 St. Paul, Baltimore 18, Secy.

Private Clinics and Hospitals Association of Texas, Dallas, Dec. 13-14, 1958. Dr. Joe Shepperd, Burnet, Pres.; Mr. Henry E. Taylor, Medical Arts Building, Dallas, Executive Secy.

Texas Academy of Internal Medicine, Galveston, Dec. 11-12, 1958. Dr. A. W. Harris, Dallas, Pres.; Dr. Hugo T. Engelhardt, 1216 Main, Houston, Secy. Meetings restricted to members.

Texas Division, American Cancer Society, Austin, Dec. 4-5, 1958. Dr. John A. Wall, Houston, Pres.; Mr. Curt W. Reimann, 5014 Bull Creek Rd., Austin 3, Executive Director.

Texas Rheumatism Association, Galveston, Dec. 5, 1958. Dr. W. W. Bondurant, San Antonio, Pres.; Dr. J. Morris Horn, 3707 Gaston, Dallas, Secy.

Texas Society of Ophthalmology and Otolaryngology, Houston, Dec. 5-6, 1958. Dr. Owen R. O'Neill, Paris, Pres.; Dr. Edwin G. Grafton, 4319 Oak Lawn, Dallas, Secy.

Texas State Board of Medical Examiners, Fort Worth, Dec. 3-6, 1958. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Secy.; Miss Luanna Knox, 1714 Medical Arts Bldg., Fort Worth, Assistant Secy.

JANUARY

Texas Radiological Society, Dallas, Jan. 30-31, 1959. Dr. R. F. Wertz, Amarillo, Pres.; Dr. J. E. Miller, 6407 Forest Lane, Dallas 30, Secy.

Texas Society of Pathologists, Houston, January 25, 1959. Dr. John H. Childers, Galveston, Pres.; Dr. Marvin H. Grossman, Box 57, Dallas, Secy.

Texas Urological Society, San Antonio, Jan. 24-25, 1959. Dr. Charles Hooks, Galveston, Pres.; Dr. Charles Hulse, 405 Medical Professional Building, San Antonio.

Twelfth District Society, Marlin, Jan. 13, 1959. Dr. W. A. Chernosky, Temple, Pres.; Dr. Bernard Roser, Corsicana, Secy.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 26-28, 1959. Dr. Lawrence B. Reppert, 817 South Texas Bldg., San Antonio, Secy; S. E. Cockrell, 202 W. French Place, San Antonio, Exec. Secy.

North Texas-Southern Oklahoma Fall Clinical Conference, January, 1959. Dr. Frank J. Lee, 1300 8th, Wichita Falls, Chm.

National and Regional

American Academy of Allergy, Chicago, Feb. 9-11, 1959. Dr. Max Samter, Oak Park, Ill., Pres.; Dr. Bram Rose, Royal Victoria Hospital, Montreal, Canada, Secy.

American Academy of General Practice, San Francisco, April 6-9, 1959. Dr. Holland T. Jackson, Fort Worth, Pres.; Mr. Mac F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Executive Secy.

American Academy of Ophthalmology and Otolaryngology. Dr. LeRoy A. Schall, Boston, Pres.; Dr. W. L. Benedict, 15 Second St. S.W., Rochester, Minn., Secy.

American Academy of Pediatrics, Chicago, Oct. 20-23, 1959. Dr. Stewart H. Clifford, Brookline, Mass., Pres.; Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill., Secy.

American Association for Maternal and Infant Health, 1960. Mr. Howard I. Wells, Jr., 116 S. Michigan Ave., Chicago 3, Executive Secy.

American Association for Thoracic Surgery, Los Angeles, April 21-23, 1959. Dr. Michael E. DeBakey, Houston, Pres.; Dr. Hiram T. Langston, 7730 Corondelet Ave., St. Louis 5, Secy.

American Association of Genito-Urinary Surgeons, Absecon, N. J., April 15-17, 1959. Dr. John A. Taylor, New York, Pres.; Dr. W. J. Engel, 2020 E. 93rd St., Cleveland 6, Secy.

American Association of Obstetricians and Gynecologists. Dr. William F. Mengert, Chicago, Pres.; Dr. E. Stewart Taylor, University of Colorado School of Medicine, 4200 E. 9th, Denver, Secy.

American Cancer Society. Dr. L. T. Coggeshall, Chicago, Pres.; M. R. Runyon, 521 W. 57th, New York, Executive Vice-Pres.

American College of Allergists, San Francisco, March 15-20, 1959. Dr. Merle W. Moore, Portland, Ore., Pres.; Dr. M. Coleman Harris, 450 Sutter St., San Francisco, Secy.

American College of Chest Physicians, Atlantic City, June 3-7, 1959. Dr. Donald R. McKay, Buffalo, Pres.; Mr. Murray Kornfeld, 112 E. Chestnut, Chicago 11, Executive Secy.

American College of Gastroenterology. Dr. C. Wilmer Wirts, Philadelphia, Pres.; Mr. Daniel Weiss, 33 West 60th, New York 23, Executive Director.

American College of Obstetricians and Gynecologists, Atlantic City, April 5-9, 1959. Dr. R. Glen Craig, San Francisco, Pres.; Dr. John C. Ullery, 15 S. Clark St., Chicago 3, Secy.

American College of Physicians, Chicago, April 20-24, 1959. Dr. Dwight L. Wilbur, San Francisco, Pres.; Mr. E. R. Loveland, 4200 Pine, Philadelphia 4, Secy.

American College of Radiology, Chicago, Feb. 6-7, 1959. Dr. Vincent W. Archer, Charlottesville, Va., Pres.; Mr. W. C. Stronach, 20 N. Wacker Dr., Chicago 6, Executive Director.

American College of Surgeons. Dr. William L. Estes, Bethlehem, Pa., Pres.; Dr. Michael L. Mason, 40 E. Erie, Chicago 11, Secy.

American Congress of Physical Medicine and Rehabilitation, Minneapolis, Aug. 30-Sept. 4, 1959. Dr. Donald L. Rose, Kansas City, Kan., Pres.; Frances Baker, One Tilton, San Mateo, Calif., Secy.

American Dermatological Association, Atlantic City, June 1-4, 1959. Dr. J. Lamar Callaway, Durham, N. C., Pres.; Dr. Wiley M. Sams, 25 Southeast 2nd Ave., Miami, Secy.

American Gastroenterological Association, Atlantic City, June 5-6, 1959. Dr. C. J. Barborka, Chicago, Pres.; Dr. F. J. Ingelfinger, 65 E. Newton, Boston 18, Secy.

American Gynecological Society, Hot Springs, Va., May 25-27, 1959. Dr. Lewis C. Scheffey, Philadelphia, Pres.; Dr. A. A. Marchetti, 3800 Reservoir Rd. N.W., Washington 7, D. C., Secy.

American Heart Association. Dr. Robert W. Wilkins, New York, Pres.; Mr. John D. Brundage, 44 E. 23rd, New York 10, Secy.

American Hospital Association, New York, Aug. 24-27, 1959. Mr. Tol Terrell, San Angelo, Pres.; Dr. Edwin L. Crosby, 18 E. Division, Chicago 10, Executive Director.

American Laryngological, Rhinological, and Otological Society, Hot Springs, Va., March 10-12, 1959. Dr. G. D. Hoople, Syracuse, N. Y., Pres.; Dr. C. S. Nash, 708 Medical Arts Bldg., Rochester, N. Y., Secy.

American Neurological Association, Atlantic City, June 15-17, 1959. Dr. Bernard J. Alpers, Philadelphia, Pres.; Dr. Charles Rupp, 133 S. 36th, Philadelphia 4, Secy.

American Ophthalmological Society, Hot Springs, Va., May 28-30, 1959. Dr. Derrick Vail, Chicago, Pres.; Dr. M. C. Wheeler, 30 W. 59th, New York 19, Secy.

American Orthopaedic Association, Lake Placid, N. Y., June 15-18, 1959. Dr. C. Leslie Mitchell, Detroit, Pres.; Dr. Lee Ramsay Straub, 535 East 70th St., New York 21, Secy.

American Pediatric Society, Buck Hill Falls, Pa., May 6-8, 1959. Dr. Joseph Stokes, Jr., Philadelphia, Pres.; Dr. A. C. McGuinness, Room 1036, 2800 Quebec St., N.W., Washington 8, D. C., Secy.

American Proctologic Society, Atlantic City, June 15-18, 1959. Dr. Karl Zimmerman, Pittsburgh, Pres.; Dr. Norman D. Nigro, 10 Peterboro, Detroit 1, Secy.

American Psychiatric Association, Philadelphia, April 26-May 1, 1959. Dr. Francis J. Gerty, Chicago, Pres.; Dr. C. H. Hardin Branch, 156 Westminister Ave., Salt Lake City, Secy.

American Public Health Association. Roy J. Morton, Oak Ridge, Tenn., Pres.; Dr. Berwyn F. Mattison, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists. Dr. Ralph S. Sappenfield, Miami 37, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph, Chicago 1, Secy.

American Society of Clinical Pathologists. Dr. Harry P. Smith, New York, Pres.; Dr. Clyde G. Culbertson, 1040 W. Michigan, Indianapolis 6, Secy.

American Surgical Association, San Francisco, April 15-17, 1959. Dr. I. S. Ravdin, Philadelphia, Pres.; Dr. William Altemeier, Cincinnati General Hospital, Cincinnati 29, Secy.

American Urological Association, Atlantic City, April 20-23, 1959. Dr. Adolph Kutzmann, Los Angeles, Pres.; Dr. Samuel L. Raines, 188 S. Bellevue Blvd., Memphis, Tenn., Secy.

Association of American Physicians and Surgeons, Fort Worth, April 2-4, 1959. Dr. Mal Rumph, Fort Worth, Pres.; Mr. Harry E. Northam, 185 N. Wabash Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter, Miami Beach, Jan. 4-7, 1959. Dr. Curtice Rosser, Dallas, Pres.; Dr. Karl Meyer, 1516 Lake Shore Dr., Chicago, Secy.

National Tuberculosis Association, Chicago, May 24-29, 1959. Dr. Mario M. Fischer, New York, Pres.; Mrs. Wallace B. White, 1790 Broadway, New York 19, Secy.

Radiological Society of North America. Dr. Leo G. Rigler, Los Angeles, Pres.; Dr. Donald S. Childs, 713 E. Genesee, Syracuse 2, N Y., Secy.

Southern Medical Association. Dr. W. Kelly West, Oklahoma City, Pres.; Mr. V. O. Foster, 1020 Empire Bldg., Birmingham 3, Secy.

Southern Psychiatric Association. Dr. Frank Luton, Nashville, Pres.; Dr. Iverson O. Brownell, Greenville, S. C., Secy.

Southwest Allergy Forum, Houston, April 26-28, 1959. Dr. Richard L. Etter, Houston, Pres.; Dr. Richard H. Jackson, 156 Hermann Professional Building, Houston, Secy.

Southwest Regional Cancer Conference. Dr. O. J. Wollenman, Fort Worth, Chm.; Mrs. Ira Frances Ball, 264 W. 11th, Fort Worth, Secy.

Southwestern Medical Association, Roswell, N. Mex., Oct.-Nov., 1959. Dr. A. R. Clausen, Albuquerque, Pres.; Dr. M. D. Thomas, 1501 Arizona St., 12-D, El Paso, Secy.

Southwestern Society of Nuclear Medicine, New Orleans, March, 1959. Dr. Herbert C. Allen, Jr., Houston, Pres.; Dr. J. R. Maxfield, Jr., 2711 Oak Lawn Avenue, Dallas, Secy.

Southwestern Surgical Congress, Denver, March 30-April 2, 1959. Dr. Louis M. Overton, Albuquerque, Pres.; Mary O'Leary, 813 Medical Arts Bldg., Oklahoma City, Exec. Secy.

Tri-State Medical Assembly. Dr. Jack Guthrie, Camden, Ark., Pres.; Dr. Jason Sanders, Sanders Clinic, Kings Highway, Shreveport, La., Secy.

United States-Mexico Border Public Health Association, Brownsville and Matamoros, Tamaulipas, Mexico, March 30-April 3, 1959. Albert O. Irigoyen, Mexico, D.F., Pres.; Dr. Jorge Román, 243 United States Court House, El Paso, Secy.

State

South Central Association of Blood Banks, Austin, February, 1959. Dr. Louis J. Manhoff, Jr., San Antonio, Pres.; L. Ruth Guy, Ph.D., Room 1101, Stoneleigh Hotel, Dallas, Secy.

Texas Academy of General Practice, Galveston, Oct. 4-7, 1959. Dr. Charles E. Oswalt, Jr., Fort Stockton, Pres.; Mr. Donald C. Jackson, 1905 N. Lamar, Austin, Executive Secy.

Texas Air-Medics Association, San Antonio, April, 1959. Dr. R. Henry Harrison, Bryan, Pres.; Dr. C. F. Miller, P. O. Box 1338, Waco, Secy.

Texas Association for Mental Health, Fort Worth, March 12-14, 1959. Dr. Frank Schooner, Fort Worth, Pres.; Mr. John Lane, 2410 San Antonio, Austin, Executive Director.

Texas Association of Obstetricians and Gynecologists, Fort Worth, Feb. 14, 1959. Dr. Jesson L. Stowe, El Paso, Pres.; Dr. Oran V. Prejean, 1317 N. Washington St., Dallas, Secy.

Texas Chapter, American Association of Public Health Physicians, San Antonio, April 19, 1959. Dr. David M. Cowgill, San Benito, Pres.; Dr. B. M. Primer, 2709 Rio Grande, Austin.

Texas Chapter, American College of Chest Physicians, San Antonio, April 19, 1959. Dr. J. O. Armstrong, Dallas, Pres.; Dr. Carlos J. Quintanilla,, Box 592, Harlingen, Secy.

Texas Club of Internists. Dr. D. D. Warren, Waco, Pres.; Dr. L. C. Carter, 2600 Procter, Port Arthur, Secy.

Texas Dermatological Society, San Antonio, April 20, 1959. Dr. Earl L. Loftis, Dallas, Pres.; Dr. E. N. Walsh, 1410 Pruitt St., Fort Worth 4, Secy.

Texas Diabetes Association, San Antonio, April 19, 1959. Dr. Merton Minter, San Antonio, Pres.; Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Secy.

Texas Geriatrics Society, Austin, Nov. 13-15, 1958. Dr. Martin S. Buehler, Dallas, Pres.; Dr. J. O. Armstrong, 3810 Swiss Ave., Dallas, Secy.

Texas Heart Association, San Antonio, April 19, 1959. Dr. William B. Adamson, Abilene, Pres.; Ernest T. Guy, 404 Jesse H. Jones Library Bldg., Texas Medical Center, Houston 25, Executive Director.

Texas Hospital Association, Houston, May 11-14, 1959. Mr. W. P. Earngey, Jr., Fort Worth, Pres.; Mr. O. Ray Hurst, 2208 Main, Dallas, Executive Director.

Texas Industrial Medical Association, San Antonio, April 19, 1959. Dr. Wendell H. Hamrick, Houston, Pres.; Dr. Robert A. Wise, Box 2180, Houston, Secy.

Texas Neurophychiatric Association, San Antonio, April 19, 1959. Dr. Samuel R. Snodgrass, Galveston, Pres.; Dr. Clarence S. Hoekstra, 8215 Westchester Drive, Dallas, Secy.

Texas Ophthalmological Association, San Antonio, April 19, 1959. Dr. Harold Beasley, Fort Worth, Pres.; Dr. James Scruggs, 2223 Austin, Waco, Secy.

Texas Orthopedic Association, San Antonio, April 20, 1959. Dr. Louis J. Levy, Fort Worth, Pres.; Dr. Margaret Watkins, 3503 Fairmount, Dallas, Secy.

Texas Pediatric Society, Fort Worth, Houston, 1959. Dr. Bruce A. Knickerbocker, Dallas, Pres.; Dr. James N. Walker, 5216 W. Freeway, Fort Worth, Secy.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 19, 1959. Dr. Vann S. Taylor, Dallas, Pres.; Dr. Lewis A. Leavitt, Veterans Administration Hospital, Houston, Secy.

Texas Proctologic Society, New Orleans, February 28, 1959. Dr. C. P. Hardwicke, Austin, Pres.; Dr. H. Gray Carter, 915 St. Joseph St., Dallas, Secy.

Texas Public Health Association, San Antonio, Feb. 22-25, 1959. Dr. D. R. Reilly, San Angelo, Pres.; Mr. Joseph N. Murphy, Jr., Box 4012, Austin 51, Executive Secy.

Texas Society of Anesthesiologists, San Antonio, April 19, 1959. Dr. M. M. Rosenzweig, San Antonio, Pres.; Dr. M. T. Jenkins, Parkland Hospital, Dallas, Secy.

Texas Society of Athletic Team Physicians, San Antonio, April 18. 1959. Dr. Edward T. Smith, Houston, Pres.; Dr. Jack Brannon, 2715 Fannin, Houston, Secy.

Texas Society of Gastroenterologists and Proctologists, San Antonio, April 19, 1959. Dr. O. P. Griffin, 800 5th Avenue, Fort Worth, Pres.; Dr. A. C. Broders, Jr., Scott and White Clinic Hospital, Temple, Secy.

Texas Society of Plastic Surgeons, San Antonio, April 18, 1959. Dr. Baron Hardy, Houston, Pres.; Dr. Raymond Brauer, 6615 Travis, Houston, Secy.

Texas Surgical Society, Austin, April 5-7, 1959. Dr. Albert Hartman, San Antonio, Pres.; Dr. G. V. Brindley, Jr., Scott and White Clinic, Temple, Secy.

Texas Traumatic Surgical Society, San Antonio, April 19, 1959. Dr. G. V. Brindley, Temple, Pres.; Dr. W. E. Crump, 1300 Eighth, Wichita Falls, Secy.

Texas Tuberculosis Association, San Antonio, March 19-21, 1959. Dr. John W. Middleton, Galveston, Pres.; Miss Pansy Nichols, P. O. Box 6158, Austin 21, Executive Director.

District

First District Society, Fort Stockton, February, 1959. Dr. H. D. Garrett, El Paso, Pres.; Dr. E. S. Crossett, 1501 Arizona St., El Paso, Secy.

Second District Society, Lamesa. Dr. J. Vernon McKay, Lamesa, Pres.; Dr. Noble L. Rumbo, Box D, O'Donnell, Secy.

Third District Society, Plainview, April 4, 1959. Dr. Robert H. Mitchell, Plainview, Pres.; Dr. H. Fred Johnson, 2308 W. Eighth, Amarillo, Secy.

Fourth District Society. Dr. W. L. Smith, San Angelo, Pres.; Dr. Braswell S. Locker, 1501 11th St., Brownwood, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1959. Dr. O. L. Riley, Corpus Christi, Pres.; Dr. Thelma Frank, 1314 16th St., Corpus Christi, Secy.

Seventh District Society. Dr. Robert N. Snider, Austin, Pres.; Dr. Richard Lucas, 502 W. 13, Austin, Secy.

Eighth District Society. Dr. John H. Childers, Galveston, Pres.; Dr. M. Warren Hardwick, 829 E. Mulberry, Angleton, Secy.

Ninth District Society, Huntsville, March 19, 1959. Dr. Lyman C. Blair, Houston, Pres.; Dr. James H. Sammons, Highlands, Secy.

Tenth District Society. Dr. B. F. Pace, Beaumont, Pres.; Dr. W. J. Posharaske, Silsbee, Secy.

Eleventh District Society, May, 1959. Dr. Ben Wilson, Tyler, Pres.; Dr. Phillip W. Taylor, 833 S. Beckham, Tyler, Secy.

Thirteenth District Society, Wichita Falls, 1959. Dr. W. R. Sibley, Abilene, Pres.; Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Secy.

Fifteenth District Society. Dr. Charles Wise, Naples, Pres.; Dr. George Bennett, 402 S. Bolivar, Marshall, Secy.

Clinics

Blackford Memorial Cancer Lectures. Dr. R. G. Gerard, 509 S. Mirick, Denison, Chm.

Dallas Southern Clinical Society, Dallas, March 23-25, 1959. Dr. C. D. Bussey, Dallas, Pres.; Millard J. Heath, 433 Medical Arts Bldg., Dallas 1, Executive Officer.

New Orleans Graduate Medical Assembly, New Orleans, March 2-5, 1959. Dr. Eugene H. Countiss, New Orleans, Pres.; Dr. Maurice E. St. Martin, 1430 Tulane Ave., New Orleans 12, Secy.

Oklahoma City Clinical Society Conference. Dr. Herman Fagin, Oklahoma City, Pres.; Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Postgraduate Medical Assembly of South Texas, Houston, July 20-22, 1959. Dr. Herbert H. Harris, Houston, Pres.; Dr. C. Forrest Jorns, Secy., Exec. Office, 412 Jesse Jones Library Bldg., Houston 25.

Board Examinations

Texas State Board of Examiners in Basic Sciences, April, 1959. Henry B. Hardt, Ph.D., Fort Worth, Pres.; Mrs. Betty J. Anderson, Chief Clerk, 303 East Seventh, Austin.

MEDICOLEGAL NOTES

Responsibility for Newborn Is Important Question

On numerous occasions questions have been raised as to who is legally responsible for the care of newborn infants in varying types of situations. From a legal standpoint, this presents an important question because when there is a legal obligation upon a physician to care and treat a patient, failure to provide such care and treatment may result in liability on the physician's part, if as a result of such "abandonment" the patient suffers injury.

Let us first take, for example, a situation in which a physician has been caring for a patient during pregnancy and it is understood that such physician is to deliver the child. Nothing is said by either party up to and including the time of the delivery about the care of the infant. In a situation of this nature there is no question but that the

physician has the legal obligation and responsibility not only to render the necessary medical services for the mother, but also to render them to the child. This responsibility to care for the child certainly would extend through the period that the mother and child were in the hospital and in all likelihood would be deemed to continue so long as the mother was under the care of the physician for reasons incident to the delivery.

If the physician, however, wishes to limit his responsibility or obligation for the care of the child, then such position should be explained to the mother well in advance of the delivery so that she will have adequate opportunity to obtain a physician to care for the child. A physician has the legal right to restrict or limit the medical services he will render, but this fact should be clearly understood and agreed to by the patient. Therefore, in a situation such as set forth, unless there is an agreement to the contrary, when a physician accepts a maternity case and then subsequently delivers the child, his responsibility does not cease with the delivery and is not limited to the care and treatment of the mother. His responsibility extends to the care and treatment of the infant or infants delivered.

Let us next take a situation in which at either the physician's or the parents' request a pediatrician is called in to care for the infant. In this situation, the question is raised as to when the delivering physician's responsibility for the care of the child ends and that of the pediatrician commences. The mere request to the pediatrician to take over the care and treatment of the child and his acceptance does not automatically end the delivering physician's responsibility to care for the child. Until the pediatrician in fact has taken over the care of the child, the delivering physician must continue to care for the medical needs of the child. Although this period between the pediatrician accepting the child as a patient and his actual taking over the care in most cases will be short in duration, the delivering physician has the obligation, during this period, to continue to care for the child.

Another situation which frequently gives rise to questions concerning upon whom the legal responsibility of caring for the child rests is one in which it is understood in advance of the delivery that a certain pediatrician is to care for the infant. This poses the question of what obligation, if any, for medical care is owed by the delivering physician to the child.

It is my opinion that even though it is understood by all parties that a certain pediatrician is to care for the child, the delivering physician has the legal responsibility to care for the child's immediate medical needs until such time as the pediatrician has been notified and has actually taken charge of the care and treatment of the child. Of course, this period is usually brief, but I believe that the delivering physician may be risking liability for "abandonment" should he fail to care for the child between delivery and the pediatrician's assumption of responsibility.

Certainly it is the pediatrician's legal responsibility, once he has accepted the request to care for the child, to make his medical services available as soon after notification of the delivery as possible. Failure to do so may subject the pediatrician to liability for "abandonment" if injury to the child can be attributed to such "abandonment."

In situations of the nature discussed here, where there is sometimes question as to whom the legal responsibility rests upon to provide the necessary medical care to the newborn infant, it is a safe course to follow, from a legal standpoint, to assume that this responsibility rests upon the delivering physician until another physician in fact has taken over the care of the child.

—PHILIP R. OVERTON, LL.B., Austin.

EDUCATION

AAPS Essay Contest Receives Favorable Publicity

A total of $2,675 in national prizes in addition to state and local prizes will be awarded winners of the Association of American Physicians and Surgeons contest on "The Advantages of Private Medical Care." The contest starts January 1, and essays must be submitted on or before March 1.

The Texas Medical Association has donated $500 for state prizes; first prize, $150, plus $100 to home room or student council of student winning first prize; second, $100; third, $75; fourth, $50; and fifth, $25. In addition, many county medical societies are offering prizes.

High school students from all public, parochial, and private schools located in the United States and its territories are eligible to enter the contest, except sons and daughters of physicians.

State judges will be former governor Dan Moody of Austin, former land commissioner Earl Rudder of College Station, and former president of Texas Medical Association, Dr. Denton Kerr of Houston. Several chambers of commerce have agreed to help.

"The Association of American Physicians and Surgeons in cooperation with state and county medical societies will do the country a great service in continuing these contests," according to Governor Moody. J. W. Edgar, Ph.D., commissioner of education in Texas, commented to officials of the association, ". . . your contest assumes a special importance in that it provides teachers an opportunity to encourage youngsters in the study of advantages of the American Free Enterprise System. Our public school teaching profession is dedicated to a perpetuation of this system and I know that teachers will welcome the stimulation and assistance you are providing to them through your contest."

Editors of the Sheridan (Wyo.) *Press* in an editorial last April commended the principles for which the AAPS contest stands. The editorial stated that the contest had grown out of concern of doctors and laymen who feared socialized medicine would undermine the private medical profession and be a major step toward national socialization.

Inquiries regarding the contest or further information may be received by writing to Dr. C. Hal McCuistion, 609 Capitol National Bank Building, Austin, state chairman for the contest.

University of Texas Medical Branch

Dr. Hans A. Krebs, director of the Department of Biochemistry at Oxford University, Oxford, England, gave the first Kempner Commemorative Lecture entitled, "The Regulation of Metabolic Processes," recently at the University of Texas Medical Branch, Galveston. Dr. Krebs received the Nobel Prize in 1953 for his discovery of what is now called the Krebs cycle.

Other visiting speakers at the Medical Branch in the past few months have been Professor Kenneth M. Smith of Cambridge University, Cambridge, England, who spoke on "Electron Microscopy of Viruses," and Professor Francisco A. Saez of Montevideo, Uraguay, who spoke on "Some Recent Investigations on Nuclear Cytology."

A testimonial dinner for Dr. and Mrs. Samuel R. Snodgrass was given by residents and former residents in neurosurgery in Galveston recently. Dr. Truman G. Blocker, Jr.,

was master of ceremonies. Residents now in training performed a skit.

Dr. R. D. Higginbotham has joined the Department of Microbiology at the Medical Branch as assistant professor. He is formerly of the University of Utah.

Postgraduate Courses

University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, January.—A course on physical and medical aspects of radioisotope use will be given Monday through Friday from 8 a.m. until 5 p.m. January 5-30. Beginning January 5 and ending February 27 is a course in radiological physics. The course will be taught Monday through Friday from 8 a.m. until 5 p.m. A course in regional anatomy will be taught from January 8 until August. Sessions will be held each Thursday from 4 to 6 p.m. Additional material on these courses may be obtained by writing the Office of the Dean, University of Texas Postgraduate School of Medicine, Texas Medical Center, Houston 25.

Nasal Surgery, Los Angeles, January 6-16.—The College of Medical Evangelists and the University of Southern California School of Medicine jointly will present a course in "Reconstructive Surgery of the Nasal Septum and External Nasal Pyramid" under the guest direction of Dr. Maurice H. Cottle, professor of otolaryngology at Chicago Medical School. Special emphasis will be placed on the newer concepts of nasal anatomy, embryology, and physiology. For further information, physicians may write Dr. Leland House, 435 South Soto Street, Los Angeles 53.

Clinical Bedside Courses, Houston, January 6-27.—A series of advanced clinical bedside courses will be presented by the Texas Medical Center Institutions in Houston. The courses are scheduled for each Tuesday from 7 to 9 p.m. Correspondence for this series is handled through the Office of the Dean, University of Texas Postgraduate School of Medicine, Texas Medical Center, Houston 25.

Internal Medicine, Houston, January 6-May 19.—Beginning January 6 on each Tuesday from 7:30 to 9:30 p.m., a course in internal medicine will be presented at St. Joseph's Hospital. Applications and other information may be obtained from the Office of the Dean, University of Texas Postgraduate School of Medicine, Texas Medical Center, Houston 25.

Internal Medicine (Therapeutics), Chicago, January 12-16.—The American College of Physicians will present a course in internal medicine, with emphasis on therapeutics, at the University of Illinois College of Medicine. Director for the course is Dr. Harry F. Dowling. Fee for fellows of the American College of Physicians is $30; $60 will be charged nonmembers. There will be sessions dealing with the use of antibiotics, steroids, and antihypertensive drugs and diuretics, and on the subjects of nephritis, psychotherapy, congestive heart failure, epilepsy, hepatic failure, diabetes, arthritis, and leukemia. E. R. Loveland, executive secretary, American College of Physicians, 4200 Pine Street, Philadelphia 4, is handling registration.

Pediatrics, Oklahoma City, January 14.—Afternoon and evening sessions on the "Diagnosis and Management of Heart Disease in Infancy and Childhood" will be held at the University of Oklahoma Medical Center. Sessions will begin at 3:30 and 8:30 p.m. More detailed information may be obtained from the Office of Postgraduate Education, University of Oklahoma Medical Center, 801 N.E. 13th Street, Oklahoma City.

Neuro-Ophthalmology, New York, January 19-23.—This 5 day course, under the direction of Dr. Alfred Kestenbaum, consists of a review of the anatomy of the nerve fibers, instrumental and noninstrumental perimetry, classification and significance of field defects, types of oculomotor and optic atrophy, chiasmal syndromes, and pupillary reactions. A short review of cerebro-ocular diseases and functional disturbances of the eye will be given. Tuition is $55. Applicants may write the Office of the Associate Dean, New York University Post-Graduate Medical School, 550 First Avenue, New York 16.

General Practice, Denver, January 19-24.—A course designed especially for the general practitioner will be presented at the University of Colorado Medical Center. Areas of practice which will be covered include medicine, pediatrics, surgery, psychiatry, obstetrics and gynecology, and trauma and dermatology. Physicians may register for the entire course or for any selected areas. The Office of Postgraduate Medical Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20, is handling program details.

Diabetes, St. Louis, January 21-23.—The American Diabetes Association is presenting a course on diabetes and basic metabolic problems in cooperation with St. Louis University School of Medicine, Washington University School of Medicine, and University of Missouri School of Medicine. Physicians desiring additional information may write the American Diabetes Association, 1 East 45th Street, New York 17.

MEDICAL MEETINGS

College of Surgeons To Meet in Houston

The American College of Surgeons will hold a 3 day sectional meeting in Houston, February 2-4, for all members of the medical profession. Reports in general surgery, a full day's otolaryngology program, a cancer workshop, films, and hospital clinics will comprise the meeting.

General sessions will include discussions on tumors, varicose veins, preparation of parents for parenthood, cancer, radiation hazards, diseases of the pancreas, and gastrointestinal tract bleeding.

The scientific program will include many speakers from Texas and a number of out-of-state guest speakers, including Dr. H. Mason Morfit, Denver; Dr. Howard Mahorner, New Orleans; Dr. Thomas H. Burford, St. Louis; Dr. William P. Longmire, Jr., Los Angeles; Dr. Curtis P. Artz, Jackson; Dr. Wiley F. Barker, Los Angeles; and Dr. James W. McLaurin, New Orleans.

Sightseeing trips and other social attractions will be available for wives of visiting surgeons. There is a $5 registration fee for doctors who attend the meeting; surgical residents, fellows of the college, and endorsed members of the candidate group of the college will be admitted free. Further information may be obtained from the American College of Surgeons, 40 East Erie Street, Chicago 11.

Dr. J. Griffin Heard, Houston, is chairman of the advisory committee on local arrangements for the meeting. Dr. R. Lee Clark, Jr., chairman of the executive committee of the Committee on Cancer, will be in charge of a cancer workshop for medical directors of approved cancer programs in Southwestern United States. The college has five governors from Texas who are Dr. Albert W. Hartman, Jr., San Antonio; Dr. Robert M. Moore, Galveston; Dr. John Paul North, Dallas; Dr. Harry M. Spence, Dallas; and Dr. George Waldron, Houston.

Texas Society on Aging Holds First Meeting

Herbert Shore of Dallas was chosen as first president of the new Texas Society on Aging, a product of a merger between the Texas Geriatric Society and the Texas Gerontological Society, at its meeting November 14-15 in Austin.

Other officers elected were Dr. Ernest Kiel, Temple, president-elect; Hiram J. Friedsam, Ph.D., Denton, vice-president; and Mrs. William B. Ruggles, Dallas, secretary-treasurer.

The new society, which carries as its slogan "Aging Is Everybody's Business," has a membership of 600 and much of its work is in relation to the Texas Interim Legislative Committee on Aging. It is a chapter of the National Gerontological Society, Inc., and is a nonpolitical, nonprofit scientific society.

The keynote speaker of the meeting, William C. Fitch, director of the special staff on aging of the United States Department of Health, Education, and Welfare, described the plans for the White House Conference on Aging to be held in January, 1961. Representative James A. Turman of Gober, vice-chairman of the Interim Legislative Committee, addressed the conference on "Recent Legislative Developments and Aging."

Physicians which have been elected to the Board of Governors are Dr. Elizabeth Gentry, Austin; Dr. Fred Dorsey, Houston; and Dr. J. O. Holt, Dallas.

Eye, Ear, Nose and Throat Meeting Held in Houston

The Texas Society of Ophthalmology and Otolaryngology held its thirty-third session, December 5-6, at the Shamrock Hilton Hotel in Houston.

Texas doctors participating in the ophthalmology sessions were Dr. James H. Scruggs, Jr., Waco; Dr. Max Baldridge, Texarkana; Dr. G. William Burch, Houston; Dr. Oscar Marchman, Jr., Dallas; Dr. Tom M. McCrory, Dallas; Dr. Charles E. Dobbs, Houston; Dr. Edward D. McKay, Amarillo; Dr. Weldon O. Murphy, Amarillo; and Dr. J. L. Mims, Jr., San Antonio. Guest speakers participating are Brigadier General Victor A. Byrnes, Washington, D. C., and Dr. Georgiana Theohold, Oak Park, Ill.

Among those taking part in the otolaryngology program were Dr. James C. Whittington, Eastland; Dr. Claude D. Winborn, Dallas; Dr. Ludwig A. Michael, Dallas; Dr. J. Mathew Robison, Houston; and Dr. Lyle M. Sellers, Dallas. Out-of-state speakers included Dr. Paul H. Holinger, Chicago, and Dr. Jerome A. Hilger, St. Paul, Minn.

Operating Room Nurses To Hold Annual Congress

The National Association of Operating Room Nurses will hold its sixth annual congress, February 9-12 at the Shamrock Hilton Hotel in Houston. The meeting will be open to doctors, registered nurses, nursing and medical students, and administrators of hospitals.

A panel presentation on "Radiation Protection" will be given by the staff of the University of Texas M. D. Anderson Hospital and Tumor Institute in Houston. Another panel discussion will be entitled "Intracardiac Surgery." Two telecasts will be presented from the operating room of the M. D. Anderson Hospital. One will demonstrate the insertion and handling of radium and the other will show open heart surgery by Dr. Denton Cooley, Baylor University College of Medicine, Houston.

For the complete program or further information, those interested may write Mrs. Marie Ellison, St. Luke's Episcopal and Texas Children's Hospitals, Texas Medical Center, Houston.

Texans Win Honors At Southern Meeting

Several Texas doctors won honors at the Southern Medical Association meeting in New Orleans, November 3-6, in addition to the many who participated on the program.

Dr. Milford O. Rouse, Dallas, was installed as president and Dr. Robert D. Moreton, Fort Worth, was elected vice-chairman of the council of the Association. Drs. A. C. Broders, Jr., R. R. White, W. H. Hunt, III, N. C. Hightower, Jr., J. C. Stinson, and Henry Laurens of Scott and White Clinic in Temple won second place in the scientific exhibit awards for their exhibit on "Primary Neoplasms of the Small Bowel."

An honorable mention exhibit award was given to Drs. Ralph Munslow, Richard Price, Robert Hardy, Francis O'Neill, Alvin Thaggard, and Jerome Wiesner of San Antonio for their exhibit entitled, "Cerebral Angiography—Its Diagnostic Value." Winners of golf trophies were Dr. F. P. Thomas of Houston and Dr. W. T. Jones of Baytown.

It was announced that the 1961 meeting will be held in Dallas and Fort Worth, November 6-9.

Texas Radiological Society Sets Meeting January 30-31

Five guest speakers have been chosen for the Texas Radiological Society meeting to be held in Dallas at the Statler Hilton Hotel, January 30-31. The meeting is open to all physicians.

Guest speakers will be Dr. C. Allen Good, Mayo Clinic, Rochester, Minn.; Dr. Gwilym S. Lodwick, University of Missouri, Columbia; Dr. Walter Murphy, Roswell Park Memorial Hospital, Buffalo; Dr. I. Steinberg, Cornell University, New York; and Dr. Theodore A. Tristan, University of Rochester, Rochester, N. Y.

Texans participating are Dr. John E. Ballard, San Angelo; Dr. Royal F. Wertz, Amarillo; Dr. George F. Crawford, Beaumont; Dr. A. D. Sears, Dallas; Dr. Benjamin DuBilier, Austin; Dr. Hugo F. Elmendorf, San Antonio; and Dr. Joseph E. Gardner, Corpus Christi.

A cocktail party and banquet will be held Friday, January 30. Registration for nonmembers is $15.

Cardiovascular Diseases Topic of Seminar

The sixth annual Seminar on Cardiovascular Diseases will be held February 19-21 in Jacksonville, Fla. It is sponsored by the Northeast Florida Heart Association in cooperation with the Division of Postgraduate Education of the College of Medicine of the University of Florida. The course will include recent developments in the diagnosis and treatment of cardiovascular diseases.

Information may be obtained from Dr. Daniel R. Usdin, 1628 San Marco Boulevard, Suite 7, Jacksonville 7, Fla.

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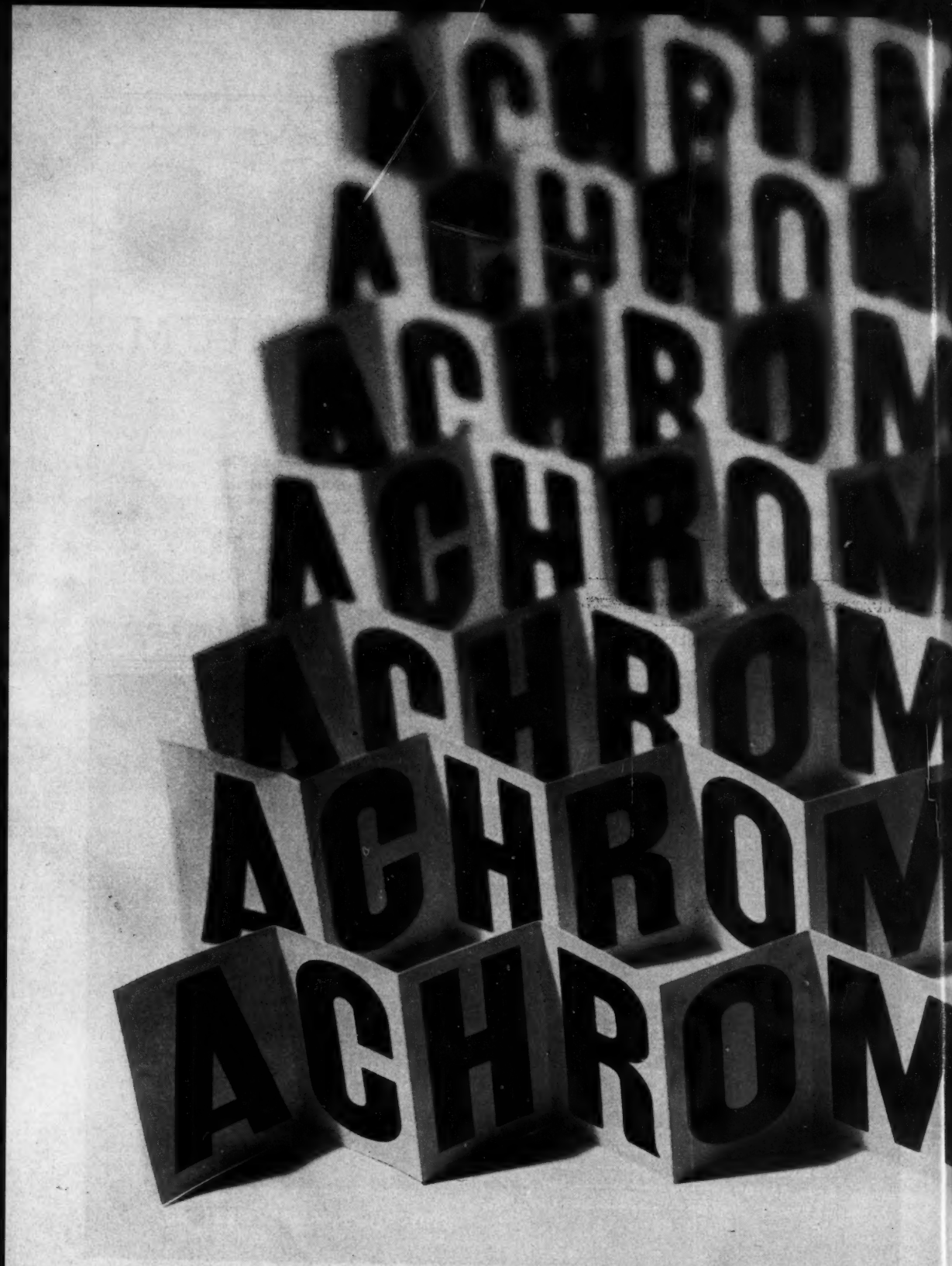
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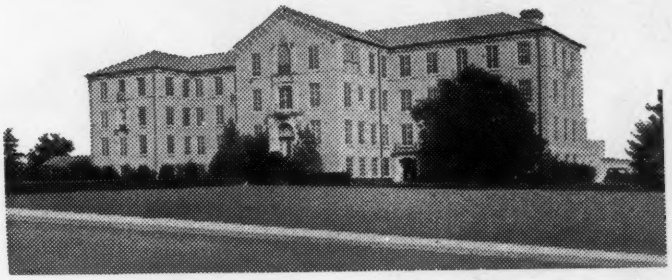
Charles L. Bloss, M.D., Medical Director
Howard M. Burkett, M.D., Associate Psychiatrist
James K. Peden, M.D., Associate Psychiatrist
Ward G. Dixon, M.D., Associate Psychiatrist
Jerry M. Lewis, M.D., Associate Psychiatrist
C. L. Jackson, M.D., Associate Psychiatrist
LeeOwen S. Buford, M.D., Associate Psychiatrist
Albert F. Riedel, Jr., M.D., Resident Psychiatrist
Belvin A. Simmons, M.D., Resident Psychiatrist
E. Clay Griffith, M.D., Resident Psychiatrist

Frances Lumpkin, R.N., B.S., Director of Nurses
Donald Bertoch, M.S., Clinical Psychologist
J. D. Martin, M.A., Clinical Psychologist
Bill M. Turnage, M.S., Director of Social Service
Lois Timmins, Ed.D., Director of Recreational Therapy
Geraldine Skinner, B.S., O.T.R., Director of Occupational Therapy
Ralph M. Barnette, Jr., Business Manager



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Charles H. Wilson, M.D., F.A.C.S.
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Jack E. Maxfield, M.D., F.A.C.S.
Clyde W. Parsons, M.D.
ORAL SURGERY
R. A. Mitchell, D.D.S.
E. E. Stewart, D.D.S.
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Owen C. Berg, M.D., F.A.C.S.
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Joe D. Steed, M.D., D.A.B.R., Radiology
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O. P. Moffitt, Jr., M.D.
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Claude D. Wilson, M.D., D.A.B.R.
Joe D. Steed, M.D., D.A.B.R.
Julian H. Acker, M.D.

MEDICINE
P. K. Smith, M.D., F.A.C.P., Diagnosis & Internal Medicine
I. L. Humphrey, Jr., M.D., Diagnosis & Internal Medicine
W. B. Whiting, M.D., F.A.C.P., Cardiology
R. F. Knox, M.D., Endocrinology
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Blair P. Coleman, M.D., Diagnosis & Internal Medicine
Preston McCall, M.D., Diagnosis & Internal Medicine
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OBSTETRICS AND GYNECOLOGY
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Warren T. Kable, M.D.
Frank J. Lee, M.D.
ADMINISTRATIVE OFFICER
Bertha McNeill, Administrator

DRUG NOTES

Bradosol Used for Throat Infections; Multiple Purpose Vaccine in Offing

BRADOSOL Lozenges (Ciba) are for mouth or throat infections and irritations. They contain benzocaine, the local anesthetic, along with Bradosol bromide. The generic name of the latter compound is domiphen bromide. Domiphen bromide is a cationic surface active agent and a quaternary ammonium compound. Cationic surface active agents exhibit germicidal as well as detergent properties and generally inhibit both gram-negative and gram-positive organisms. Usually they are not effective against spores nor too effective with fungi.

Chemically, Bradosol bromide is dodecyldimethyl-(2-phenoxyethyl) ammonium bromide. Quaternary ammonium compounds may be thought of as having the four bonds (covalent) of nitrogen linked to four hydrocarbon chains. Usually one of the hydrocarbon chains is long (in this case dodecyl-, 12 carbons) and may contain an aromatic or oxygenated modification. The other substituents on the nitrogen may be short alkyl or aromatic in nature (in this case two methyl groups and one phenoxyethyl).

Thus, it can be seen that Bradosol bromide belongs to the same class of compounds as Zephiran (Winthrop), Phemerol (Parke-Davis), Ceepryn (Merrill), Diaparene, Cetavlon, and cetyldimethylethyl ammonium bromide.

Bradosol is said to be nontoxic with sensitivity a rarity. Low concentrations of the drug are said to kill most pathogenic organisms of patients with mouth and throat infections. The lozenges are also suggested for use prophylactically, such as following dental extractions and tonsillectomies. The lozenges are supplied 24 in a package about the same size and shape as that of a king-sized cigarette's and retail for about 69 cents.

QUADRIGEN (Parke-Davis), a four-in-one vaccine to immunize against polio, whooping cough, diphtheria, and tetanus may be commercially available in a few months.

Two new antibiotics among those reported at the sixth annual Antibiotic Symposium were Actinobolin (Parke-Davis) and Mitromycin (Bristol). These are still under trial and are being tested as anticancer agents.

—HERBERT SCHWARTZ, M.S., Austin.

OF GENERAL INTEREST

Personals

Dr. Carleton B. Chapman, Dallas, participated in the tenth annual Symposium on Heart Disease sponsored by the Washington State Heart Association in cooperation with the Washington State Department of Health, October 17-18, at Seattle.

Dr. Charles W. Tennison of San Antonio was elected vice-president of the American Society of Plastic and Reconstructive Surgery at the recent convention held in Chicago.

Dr. Howard O. Smith's mother, Mrs. Clara Buchanan, died in Marlin on November 15 and was buried in Hamilton, her home town, November 16. She had been living in Marlin recently.

Dr. Howard C. Coggeshall of Dallas is editor of a forthcoming issue of *Medical Clinics of North America* entitled "Common Pain Problems." *Drs. Alfred W. Harris, Charles F. Gregory,* and *Jesse E. Thompson* of Dallas are other contributing authors.

Dr. J. W. Bass, director of public health for Dallas, received a special award upon completion of 33 years of service in the Army. A colonel, Dr. Bass has gone on inactive reserve status.

Dr. Lee D. Cady, manager, Veterans Administration Hospital, Houston, was elected to the executive committee of the new Medical School-Teaching Hospital Section of the Association of American Medical Colleges. The initial meeting of the section occurred October 9-10.

Dr. Horace H. Trippet of Waco married Miss Nelwyn Green Harmon of Overton recently. They are residing in Waco where Dr. Trippet practices.

A girl, Nancy Lee, was born to *Dr. and Mrs. Livius L. Lankford* of Dallas on August 15.

Dr. and Mrs. John L. Lukowski are parents of a daughter, Suzanne Elizabeth, who was born September 21 in El Paso.

Tol Terrell, hospital administrator of San Angelo, was elected a director of the Joint Council to Improve the Health Care of the Aged on October 25. The council is sponsored by the American Dental Association, American Hospital Association, American Medical Association, and American Nursing Home Association.

At the fifteenth annual meeting of the southern chapter of the American College of Chest Physicians held in New Orleans, November 2-3, *Dr. Daniel E. Jenkins,* Houston, was elected president and *Dr. Henry R. Hoskins,* San Antonio, secretary-treasurer.

Dr. James L. Johnson of Amarillo presented the original manuscript of "The Eyes of Texas," the University of Texas alma mater, for permanent loan to the University at ceremonies preceding the Texas-Texas A. and M. football game on November 28 in Austin.

At the International College of Surgeons' Southwestern Regional Meeting of the United States Section in Miami Beach, Fla., January 4-7, *Dr. Curtice Rosser* of Dallas and *Dr. Hugh Beaton* of Fort Worth will be guest speakers.

A dinner honoring *Dr. Pat I. Nixon* of San Antonio on his seventy fifth birthday was held November 25 in San Antonio for approximately 300 guests. Frank Wardlaw, Austin, director of the University of Texas Press, which printed Dr. Nixon's book on "A History of the Texas Medical Association," spoke.

Dr. Max Johnson, San Antonio, was selected as a member of the City Council of San Antonio recently.

Dr. Denton Cooley, member of the Baylor University College of Medicine faculty, Houston, will participate in a symposium on surgical therapy of congenital heart disease for the medical sciences section of the American Association for the Advancement of Science in Washington, D. C., December 26-31.

Dr. William O. Russell, Houston, attended the meeting of the National Committee for Careers in Medical Technology, founded by the American Society of Medical Technologists and the College of American Pathologists, November 3 in Chicago. He is chairman of the committee.

Dr. Erwin O. Strassmann of Houston has been elected as one of the main speakers for the third World Congress on Fertility and Sterility to be held in Amsterdam in June of 1959. Dr. Strassmann, whose paper will be read at plenary session, the highest category of presentations, will report on his research on the mechanism of the ovulation.

Dr. Lyle M. Sellers of Dallas was elected as one of three vice-presidents of the American Academy of Ophthalmology and Otolaryngology at the annual meeting of the academy in Chicago October 16.

Texas Tuberculosis Association Celebrates Fiftieth Anniversary

The Texas Tuberculosis Association will close its golden anniversary year this month, hoping to make the 1958 Christmas seal drive a successful one, reports Dr. Elliott Mendenhall, chairman of the Committee on Tuberculosis of the Texas Medical Association and a director of the tuberculosis association.

In an appeal to Texas physicians for their support in this year's drive, Dr. Mendenhall said that for 50 years, the tuberculosis association has worked hand in hand with medical and allied groups to educate lay people concerning tuberculosis and to seek in every way possible to improve patient care and rehabilitation.

The year 1958 has brought about several changes in policy with regard to tuberculosis control, along with an effort for coordination of activities in the field. The association has cooperated in its efforts with the Texas State Department of Health, the Board for Texas State Hospitals and Special Schools, the Texas Coordinating Council on Tuberculosis, and the Texas Medical Association.

Help Fight TB

Buy Christmas Seals

The association has undertaken the education of the public to the changing methods of case findings, among which has been the more limited use of mass roentgenray screening of the population. A greater emphasis has been placed on research toward a possible vaccine for immunity and toward better methods of case findings.

Other main projects for the year have included a blood recruitment program for patients in state tuberculosis hospitals requiring surgery, periodic visits to state supported tuberculosis hospitals, encouragement of the team approach to tuberculosis through the organization of local tuberculosis councils similar to the Texas Coordinating Council on Tuberculosis, and steps toward a recodification of Texas tuberculosis laws, which have contained many conflicting, outdated, and confusing provisions.

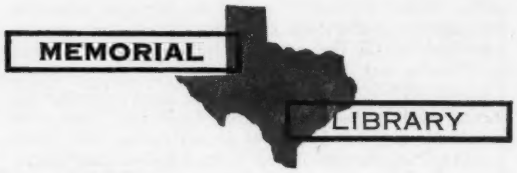
Department of Health Buildings Dedicated in Austin

Dedication ceremonies of the Texas State Department of Health laboratory and administration buildings were held November 21 in Austin.

The laboratory and administrative buildings were constructed 1956-1958 with funds appropriated by the Texas Legislature and through the federal hospital construction program.

Governor Price Daniel and Dr. Leroy Burney, surgeon general, United States Public Health Service, gave addresses. Dr. Henry A. Holle, commissioner of health, Austin, acted as master of ceremonies, with Dr. J. B. Copeland of San Antonio, chairman of the State Board of Health, also participating. The Bergstrom Air Force Base Color Guard and McCallum High School Band of Austin performed a flag ceremony. Members of the Texas State Board of Health and Department of Health of the City of Austin, as well as Dr. Howard O. Smith of Marlin, President of the Texas Medical Association, were introduced as guests.

Doctors of medicine other than Dr. Copeland currently serving on the State Board of Health are Dr. George Turner, El Paso; Dr. R. W. Kimbro, Cleburne; Dr. F. W. Yeager, Corpus Christi; and Dr. Hampton C. Robinson, Houston.



Gifts to the Library

Monetary gift by Kerr-Kendall-Gillespie-Bandera Counties Medical Society in memory of Dr. H. H. Gallatin, Kerrville.

Dr. C. H. Brownlee, Austin, 88 books and bound volumes.

Dr. Morris Davidson, Austin, 64 journals.

Dr. Morris Jacobs, Austin, 19 journals.

Dr. Samuel N. Key, Austin, 6 journals.

Dr. Heinrich Lamm, Austin, 1 book, 176 journals, 6 reprints.

Dr. Joe C. Rude, Austin, 2 journals.

W. E. Syers, Kerrville, 49 booklets, 2 journals, 4 books.

Dr. David Wade, Austin, 25 journals.

★ Books

Books Newly Acquired

Ciba Foundation, Ciba Foundation Colloquia on Aging: *Water and Electrolyte Metabolism in Relation to Age and Sex,* vol. 4, Boston, Little, Brown and Co., 1958.

Ciba Foundation Symposium, G. E. W. Wolstenholm, Ed.: *Neurological Basis of Behaviour,* Boston, Little, Brown and Co., 1958.

Cowdry, E. V., *The Care of the Geriatric Patient,* St. Louis, C. V. Mosby, 1958.

Gofman, John W., *What We Do Know About Heart Attacks,* New York, G. P. Putnam's, 1958.

Hardy, James D.: *Pathophysiology in Surgery,* Baltimore, Williams and Wilkins, 1958.

Hollinshead, W. Henry: *Anatomy for Surgeons: The Back and Limbs,* vol. 3, New York, Hoeber-Harper, 1958.

Hopkins, Edward Scott, and Schulze, Wilmer Henry: *Practice of Sanitation,* ed. 3, Baltimore, Williams and Wilkins, 1958.

Jablonski, Stanley: *Russian-English Medical Dictionary,* New York, Academic Press, 1958.

Jordan, Edwin P.: *Physician and Group Practice,* Chicago, Year Book Publishers, 1958.

Sakel, Manfred: *Epilepsy,* New York, Philosophical Library, 1958.

Sakel, Manfred: *Schizophrenia,* New York, Philosophical Library, 1958.

Saphir, Otto: *Autopsy Diagnosis and Technic*, ed. 4, New York, Hoeber-Harper, 1958.

Schneck, Jerome M.: *Studies in Scientific Hypnosis*, New York, Nervous and Mental Diseases Monographs no. 84, 1954.

Segaloff, Albert, Ed.: *Breast Cancer: Biennial Louisiana Cancer Conference*, ed. 2, St. Louis, C. V. Mosby, 1958.

Stallard, H. B.: *Eye Surgery*, ed. 3, Baltimore, Williams and Wilkins, 1958.

Von Oettingen, Wolfgang F.: *Poisoning*, ed. 2, Philadelphia, W. B. Saunders, 1958.

Wiener, A. S., and Wexler, I. B.: *Heredity of the Blood* Groups, New York, Grune and Stratton, 1958.

Book Notes

Infectious Diseases of Children

SAUL KRUGMAN, M.D., New York, Associate Professor of Pediatrics, New York University College of Medicine; and ROBERT WARD, M.D., Los Angeles, Professor and Head of the Department of Pediatrics, University of Southern California. 340 pages. $10. St. Louis, C. V. Mosby Company, 1958.

This is an excellent book which accomplished well its stated purpose of providing a concise and handy description of certain common infectious diseases of children. "It is written primarily for pediatricians, general practitioners, and medical students who deal with children."

Portions of the book are especially helpful; the chapter on adenoviruses presents a current review of the clinical syndromes associated with these agents; the chapter on herpes simplex infection describes nicely the clinical manifestation of the primary infection.

Much current information is presented in the chapter on ECHO and Coxsackie viruses describing the clinical syndrome produced by these agents including recent descriptions of the skin rashes.

The method used in presenting each illness is generally this: etiology, pathology, clinical manifestation, diagnosis, differential diagnosis, complications, treatment, prognosis, epidemiologic factors, immunity, and control measures.

Throughout its entirety this book is thorough yet concise. The presentation of each illness is followed by pertinent references.

Pediatricians and others treating the acute infectious diseases of children will find this book a gold mine of up-to-date information.

—James R. McNiel, M.D., Austin.

The Diagnosis and Treatment of Infections

D. GERAINT JAMES, M.A., M.D., (Cantab.), M.R.C.P. (Lond.), Clinical Assistant, Middlesex Hospital, London. 234 pages. $6. Springfield, Ill., Charles C Thomas, 1957.

This small book presents a well organized, concise, yet complete review of infectious diseases and the chemotherapeutic agents currently available for treating these diseases.

The coverage of the volume is not at all exhaustive, but, on the other hand, it is a good review or reference manual for the student or the busy practitioner. It is divided conveniently into three parts: "Chemotherapeutic Agents," "Micro-Organisms Causing Human Disease," and "Infections of Systems."

The author, Dr. James, is to be complimented particularly for the inclusion of 24 tables which organize the available facts into nice, compartmented, informative groups of "pearls."

—H. C. Day, M.D., Boerne.

You Can Increase Your Heart-Power

PETER J. STEINCROHN, M.D., F.A.C.P. 381 pages. $4.95. Garden City, N. Y. Doubleday and Company, 1958.

In this volume the author has discussed and answered many of the questions that arise in the minds of people concerning heart disease, hypertension, overweight, tobacco, alcohol, and other subjects related to one's health.

The author tries to school the reader on the recognition of organic heart disease and imaginary heart disease. He also tries to inform the patient about coronary disease and promote a more satisfactory doctor-patient relationship. He discusses the problem of arteriosclerosis and high blood pressure in a manner pleasing to the laymen.

The forte of this book is its discussion of numerous subjects and complaints that are known to well informed laymen and its explanation in simple terms of the answers to these problems and that they should be solved in cooperation of patient and doctor.

This book will be of greatest value to the lay public.

—Thomas R. Jones, M.D., Jasper.

Diagnostic Laboratory Hematology

GEORGE E. CARTWRIGHT, M.D., Professor of Medicine, College of Medicine, University of Utah, Salt Lake City, ed. 2. 250 pages. $6.75. New York, Grune and Stratton, Inc., 1958.

This book is a thorough but concise presentation of the procedures commonly used in student laboratories and physicians' offices for the diagnosis of hematologic disorders. In instances where numerous techniques are available for one determination the author has selected the procedure of choice based on his experience. As the preface states, "accuracy, reliability, time of performance, cost and availability of equipment are all factors which have been taken into consideration."

In addition, this second edition contains procedures for the determination of hemoglobin, heme pigments in serum, osmotic fragility and serum bilirubin which require the use of a photometer. A method for counting platelets with a phase contrast microscope and procedures for the identification of abnormal hemoglobins by paper electrophoresis are described. The equipment for electrophoresis is such that it may be assembled in a small laboratory and employs a "Heathkit" variable voltage power supply, a suggestion which effects a sizable economy.

This book will be useful to students and younger physicians who are equipping their offices.

—James R. McNiel, M.D., Austin.

Ulcers of the Legs

P. PIULACHS, Chief Professor Surgeon in the Faculty of Medicine of Barcelona; introduction by RUDOLPH MATAS, M.D., New Orleans. 574 pages. $15.50. Springfield, Ill., Charles C Thomas, 1956.

The Preservation of Youth: Essays on Health

MOSES BEN MAIMON (Maimonides). Translated from the original Arabic (Fi Tadbir As-Sihha), with an introduction by Hirsch L. Gordon, M. D., Ph. D. 92 pages. $2.75. New York, Philosophical Library, 1958.

The Nature of Viruses

Ciba Foundation Symposium, edited by G. E. W. WOLSTENHOLME, M.B., B.Ch. and ELAINE C. P. MILLAR, A.H.-W.C., A.R.I.C. 292 pages. $9. Boston, Little, Brown and Company, 1957.

Psychopathology of Communication

Edited by PAUL H. HOCH, M.D., New York State Psychiatric Institute; College of Physicians and Surgeons, Columbia University, New York, and JOSEPH ZUBIN, Ph.D., New York State Psychiatric Institute; Department of Psychology, Columbia University, New York. Proceedings of the Forty-Sixth Annual Meeting of the American Psychopathological Association, held in New York, June, 1956. 305 pages. $6.75. New York, Grune and Stratton, 1958.

★ American Medical Association

Variety of Issues Considered During AMA Clinical Meeting

The 1958 clinical session of the American Medical Association brought action from the House of Delegates on health care of the aged, medical care plans, osteopathy, expansion of medical education facilities, administration and other changes in objectives and basic programs of the association, voluntary health organization fund raising, and a variety of other issues. The meeting was held in Minneapolis December 2-5 with a registration on Thursday, December 4, of 4,880, of which 2,870 were physicians. The 1959 clinical session will be held in Dallas.

Among proposals adopted by the House were the following:

The association will expedite the development of an effective voluntary health insurance or prepayment program for persons over 65 with modest resources or low family income. Under this proposal, physicians will accept a level of compensation for medical services which will permit the development of such insurance and prepayment plans at a reduced premium rate.

The House deferred action on the report of the Commission on Medical Care Plans, suggesting that constituent associations review the report and come to definite decisions with regard to free choice of physicians and the closed panel systems.

Action regarding relations with the osteopathic profession was delayed until the June, 1959, meeting pending further review on the subject by the Judicial Council. The House, however, "noted with favor that the American Osteopathic Association has amended its objectives as stated in its constitution by deleting reference to the cultism of Andrew J. Still."

The House approved a statement by the Council on Medical Education and Hospitals supporting the development of additional facilities for basic medical education and urging the existing medical schools to consider the possibility of increasing their enrollments and developing new facilities.

A resolution on the AMA staff called for the reorganization of divisions into business, law, communications, field, scientific publications, socio-economic activities, and scientific activities.

Among the provisions presented by the Committee to Study AMA Objectives and Basic Programs which were approved by the House was one stating that the objectives of the association are to promote the science and art of medicine, the betterment of public health, and an understanding of the socio-economic conditions which will facilitate the attainment of these objectives. The promotion of an active liaison with each national medical society and consideration of the opening of the association's publications to discussion of socio-economic problems applicable to medicine were urged. Dr. Milford O. Rouse of Dallas served on the objectives committee.

The House neither approved nor disapproved the inclusion of voluntary health agencies in United Fund drives. A top-level conference with voluntary health agencies, the United Funds, and other interested parties was requested.

Regret was expressed at the substitution of federal facilities for private care in the areas mentioned in the Medicare program. The reestablishment of service under the free choice principle to accomplish the original intent of the act was urged.

A recommendation was made that the social security act be amended by Congress to permit states to combine the present four public assistance medical programs into a single medical program administered by a single agency, making available uniformity of services to all eligible public assistance recipients.

A congress on prepaid health insurance, a plan to develop "Buyers' Guides" to be sent to physicians to help patients analyze the merits of available health insurance programs, and the voluntary registration of the paramedical personnel who assist physicians were approved. The extension of governmental licensure and governmental registration of paramedical personnel was opposed.

Dr. Lonnie A. Coffin of Farmington, Iowa, was named General Practitioner of the Year.

The House expressed appreciation for the service of Dr. Austin Smith, who resigned as editor of the *Journal of the American Medical Association*, effective December 15.

Dr. J. B. Copeland of San Antonio served as chairman of the Reference Committee on Miscellaneous Business and Dr. T. C. Terrell was one of the five members of the Reference Committee on Medical Military Affairs.

Responsibility Key to Optimum Use, AMA Insurance Committee States

Health insurance constitutes only a pooling of funds from which amounts are withdrawn to pay or assist to pay the health care expenses of the individual, Dr. Carlton E. Wertz, Buffalo, N. Y., chairman of the Committee on Insurance and Prepayment Plans of the American Medical Association's Council on Medical Service, has pointed out. Consequently, it is of importance that all parties to these plans recognize and assume their respective responsibilities in order that the premium dollars may be utilized to provide optimum medical and hospital care.

The committee unanimously adopted the following statement in a September meeting:

1. The individual physician should advise hospitalization only when definitely indicated for the best care of the patient's condition and should return each patient to his home environment as soon as efficient professional care permits.

Many hospital staffs have found that an Admissions Review Committee has assisted materially in assuring a more efficient utilization of hospital facilities.

2. The subscriber should expect hospitalization only when warranted, thereby avoiding the inevitable rise in premium rates resulting from excessive utilization.

3. The insurance company or prepayment plan should clearly delineate, in the policy, the risks assumed. In the selling of these policies the subscriber should receive a complete explanation of the nature and extent of the coverage provided.

Prepayment plans, insurance companies, and, in the case of group plans, management and union officials should improve educational procedures to inform subscribers of the advantages of health insurance and dangers of increased cost by unnecessary utilization.

Efforts should be made to revise coverage so that hospitalization is not the sole requisite for obtaining some types of benefits.

4. The hospital administrators should accelerate their studies and seek to improve management practices designed to stabilize hospital costs.

★ Texas Medical Association

County Society Officials Meeting Features 5 Guest Speakers

Five guest speakers have accepted invitations for the eighth annual Conference of County Medical Society Officials and Symposium on Legislation January 24-25, at the Texas Medical Association headquarters building in Austin.

The conference will be held concurrently with the orientation program for new and transfer members on Saturday, January 24. Among the guest speakers will be Dr. Ernest B. Howard, Chicago, assistant executive vice-president, American Medical Association; George Bugbee, New York, president, Health Information Foundation; Dr. Donald E. Stubbs, Washington, D. C., chairman, Board of Trustees, Blue Shield Commission; Dr. John E. McDonald, Tulsa, member Committee on Legislation, American Medical Association; and Frank Ikard, Wichita Falls, Representative, Thirteenth Congressional District of Texas.

The Executive Council will be in session Sunday, January 25, and many of the Association's boards, councils, and committees will meet Saturday. Physicians, their ladies, and friends are invited to be guests of the Medical Protective Company for a hospitality hour Saturday at 6:30 p. m. at the Driskill Hotel.

Optional Benefits Offered Great American Policyowners

Current policyowners in the term life insurance plan offered by the Great American Reserve Insurance Company of Dallas, under sponsorship of the Texas Medical Association, are now entitled to a new optional benefit schedule, giving larger amounts of insurance at older ages. The new schedule has been approved by the Association's Council on Medical Economics.

The insurance plan previously offered calls for a single premium of $100 per year, with benefits varying from $26,000 to $1,750 based on age of the insured. Premiums on this plan do not increase as the insured grows older; the amount of insurance is reduced. The alternate plan, however, calls for a larger premium as the insured advances in age, at the same time, increasing the amount of insurance.

Premiums and benefits as set forth in the alternate plan include the following: $100 per year, $26,000 up to age 35; $100 per year, $15,000 from 36 to 45; $133.33 per year, $10,000 from 46 to 55; $214.28 per year, $7,500 from 56 to 65; and $244.95 per year, $5,000 from 66 to 70, all ages being to the nearest birthday.

Those who wish may continue the original plan, which also is available to new applicants.

Booklet on Claim Forms Sent TMA Members

A guide for physicians on the use of new simplified claim forms for accident and health insurance policies which was published by the Health Insurance Council has been sent to members of the Texas Medical Association recently.

These forms have been approved by the American Medical Association's Council on Medical Service on the recommendation of its Committee on Prepayment Medical and Hospital Service. The development of simplified claim forms for doctors to report accident and health claims may help members with the problem of paper work. These forms, however, should not be confused with the "Standard Hospital Forms," which are sponsored by the Hospitals-Insurance-Physicians Joint Advisory Committee of Texas and are now in use in Texas.

San Antonio Committees Making Plans

Arrangements for the 1959 annual session of the Texas Medical Association are being worked out by San Antonio physicians under the leadership of Dr. A. Fletcher Clark, Jr., chairman of the Committee on General Arrangements appointed by the President of the Association.

Serving with Dr. Clark as members of the state committee are the six chairmen of his local committees:

Alumni Dinners.—Dr. William M. Center, chairman (Arkansas); Dr. Thomas Hebert (Louisiana State), Dr. Dan A. Russell, Jr. (Tulane), Dr. Carl J. Walker (Tennessee), Dr. Darrell Willerson (Baylor). A committeeman for the University of Texas Medical Branch is yet to be named.

Fraternity Parties.—Dr. James L. Mims, Jr., chairman (Phi Chi); Dr. Rolan R. Botkin (Phi Rho Sigma), Dr. Royal B. Lea (Theta Kappa Psi), Dr. David T. McMahon, Jr. (Phi Beta Pi), Dr. Charles M. Manhoff (Alpha Kappa Kappa), Dr. M. M. Rosenzweig (Phi Delta Epsilon), Dr. Charles F. Skripka (Nu Sigma Nu).

Golf.—Dr. Ernest A. Maxwell, chairman; Dr. Arthur B. Pyterek, Dr. M. Bradfield Oxford.

Housing.—Dr. William D. Montgomery, chairman.

Memorial Services.—Dr. John H. Bohmfalk, chairman.

Sponsorship.—Dr. Charles L. McGehee, chairman.

Inquiries relating to the subjects of these committees may be directed to Dr. Clark, 506 Medical Professional Building, San Antonio, or to the committeemen concerned.

Conference of County Medical Society Officials and Symposium on Legislation

Texas Medical Association Headquarters Building Austin, January 24, 1959

Morning Program, 10:30 a. m.

Call to Order.—DR. C. E. WILLINGHAM, Tyler, Vice-President.

Spectatoritis.—DR. HOWARD O. SMITH, Marlin, President.

Building a Stronger and More Effective Medical Organization.—DR. ERNEST B. HOWARD, Chicago, Assistant Executive Vice-President, American Medical Association.

DR. HOWARD

Observations on Government Medicine in England.—GEORGE BUGBEE, New York, President, Health Information Foundation.

Projects for County Medical Societies in 1959:

Panel Discussion: Voluntary Health Insurance.—DR. A. REX KIRKLEY, Belton, Committee on Voluntary Health Insurance and Health Costs.

Problems of the Aging.—DR. WENDELL D. GINGRICH, Galveston, Committee on Problems of the Aging.

Closer Liaison with the Woman's Auxiliary.—MRS. WILLIAM D. NICHOLSON, Angleton, First Vice-President, Woman's Auxiliary.

The Best Medical Care for All Texas.—C. LINCOLN WILLISTON, Austin, Executive Secretary.

Luncheon, 1 p. m.
Afternoon Program, 2 p. m.

DR. MCDONALD

Call to Order.—DR. CHARLES P. HARDWICKE, Austin, Speaker of the House of Delegates.

Blue Shield and American Medicine.—DR. DONALD E. STUBBS, Washington, D. C., Chairman, Board of Directors, Blue Shield Medical Care Plan.

What's Ahead for Medicine in 1959 . . .

1. . . . *in the 86th Congress.*—DR. JOHN E. MCDONALD, Tulsa, Committee on Legislation, American Medical Association.
2. . . . *in the State Legislature.*—PHILIP R. OVERTON, Austin, General Counsel.
3. . . . *in the AMA House of Delegates.*—DR. MILFORD O. ROUSE, Dallas, Chairman, Texas Delegates to the American Medical Association.

MR. IKARD

4. . . . *in the TMA House of Delegates.*—DR. JAMES D. MURPHY, Fort Worth, Vice-Speaker, Texas Medical Association's House of Delegates.

Congressional Issues of Particular Interest to Physicians.—FRANK IKARD, Wichita Falls, Representative, Thirteenth Congressional District of Texas.

Evening Program, 6:30 p. m.

Hospitality Hour.—Driskill Hotel (Physicians, wives, and friends are invited to be guests of the Medical Protective Company).

Orientation Program

Texas Medical Association Headquarters Building

Austin, January 24, 1959

Morning Program, 9:20 a. m.

Welcome.—DR. TRAVIS SMITH, Abilene, Vice-Chairman, Board of Councilors.

The Texas Medical Association; An Accounting of Stewardship.—What Happens to Your $50.—DR. TROY A. SHAFER, Harlingen, Member, Board of Trustees.

Public Relations and Socio-Economic Considerations in the Practice of Medicine.—C. LINCOLN WILLISTON, Austin, Executive Secretary.

Spectatoritis.—DR. HOWARD O. SMITH, Marlin, President.

Building a Stronger and More Effective Medical Organization.—ERNEST B. HOWARD, Chicago, Assistant Executive Vice-President, American Medical Association.

DR. BUGBEE

Speaker to be announced.

Observations on Government Medicine in England.—GEORGE BUGBEE, New York, President, Health Information Foundation.

Luncheon, 12:30 p. m.

Afternoon Program, 1:25 p. m.

Medical Ethics Considerations in the Practice of Medicine.—DR. C. E. OSWALT, JR., Fort Stockton, Chairman, Board of Councilors.

Medical Etiquette; Obligations of the Physician to Colleagues and to the Profession.—DR. R. MAYO TENERY, Waxahachie, Councilor, District 14.

Blue Shield and American Medicine.—DR. DONALD E. STUBBS, Washington, D. C., Chairman, Board of Directors, Blue Shield Medical Care Plan.

Serving the Doctors of Texas.—DONALD M. ANDERSON, Austin, Assistant Executive Secretary.

DR. STUBBS

Workmen's Compensation Laws; Charges, Obligations, and the Law.—SMITH PETTIGREW, Dallas, Medical Coordinator, Texas Employers' Insurance Association.

Legal Aspects of Medical Practice; Malpractice—How to Avoid It.—PAT BAILEY, Austin, Assistant General Counsel.

Congressional Issues of Particular Interest to Physicians.—FRANK IKARD, Representative, Thirteenth Congressional District of Texas.

Evening Program, 6:30 p. m.

Hospitality Hour.—Driskill Hotel (Physicians, wives, and friends are invited to be guests of the Medical Protective Company).

25 Guest Speakers Obtained For Annual Session Program

A total of 25 out-of-state speakers, representing a variety of phases of medicine, have accepted invitations to participate in the Texas Medical Association annual session scheduled in San Antonio April 19-21. Speakers and the sections or specialty societies which are sponsoring them include the following:

Dr. Walter C. Alvarez, professor emeritus of medicine, University of Minnesota Medical School, Chicago, Section on General Practice.

Dr. George C. Andrews, clinical professor of dermatology (retired), College of Physicians and Surgeons, Columbia University, New York, Texas Dermatological Society.

Dr. Harry E. Bacon, professor and head, Department of Rectal and Colonic Surgery, Temple University School of Medicine, Philadelphia, Texas Society of Gastroenterology and Proctology.

Dr. Richard J. Bing, professor of medicine, Washington University School of Medicine, St. Louis, Texas Heart Association.

Dr. Edward L. Compere, professor and chairman, Department of Orthopedic Surgery, Northwestern University Medical School, Chicago, Texas Orthopedic Association.

Dr. Murray M. Copeland, professor and chairman, Department of Oncology, George Washington School of Medicine, Washington, D. C., Texas Division, American Cancer Society.

Dr. James E. Eckenhoff, associate professor of anesthesiology, University of Pennsylvania School of Medicine, Philadelphia, Texas Society of Anesthesiologists.

Dr. John B. Erich, head, department of Plastic Surgery, Mayo Clinic, Rochester, Minn., Section on Eye, Ear, Nose, and Throat.

Dr. Jack R. Ewalt, commissioner, Department of Mental Health, Commonwealth of Massachusetts, Boston, Texas Industrial Medical Association.

Dr. Grace A. Goldsmith, professor of medicine, Tulane University School of Medicine, New Orleans, Texas Diabetes Association.

Dr. Gunnar Gundersen, President, American Medical Association, La Crosse, Wis.

Dr. Eugene A. Hargrove, associate professor of psychiatry, University of North Carolina School of Medicine, Chapel Hill, Texas Neuropsychiatric Association.

Dr. Herman E. Hilleboe, commissioner of health, State of New York, Albany, Section on Public Health and Conference of City and County Health Officers.

Dr. Oscar B. Hunter, Jr., director, Oscar B. Hunter Memorial Laboratory, Washington, D. C., Section on Pathology.

Dr. Ted F. Leigh, professor of radiology, Emory University School of Medicine, Atlanta, Section on Radiology.

Dr. Champ Lyons, professor and chairman, Department of Surgery, Medical College of Alabama, Birmingham, Section on Surgery.

Dr. Donald D. Matson, neurosurgeon, Children's Hospital, Boston, Texas Neuropsychiatric Association and Texas Pediatric Society.

Dr. John M. McLean, professor of clinical surgery, Cornell University Medical College, New York, Section on Eye, Ear, Nose, and Throat and Texas Ophthalmological Association.

Dr. Newell W. Philpott, obstetrician and gynecologist, Montreal, Canada, Texas Division, American Cancer Society.

Dr. Duncan E. Reid, professor and head, Department of Obstetrics, Harvard Medical School, Boston, Section on Obstetrics and Gynecology.

Dr. Mitchell Rubin, professor and head, Department of Pediatrics, University of Buffalo School of Medicine, New York, Section on Pediatrics.

Dr. Oscar Sander, professor of occupational and environmental medicine, Marquette University School of Medicine, Milwaukee, Texas Chapter, American College of Chest Physicians.

Dr. John T. Sessions, Jr., associate professor of medicine, University of North Carolina School of Medicine, Chapel Hill, Section on Internal Medicine.

Dr. Byron Smith, associate clinical professor of ophthalmology, New York University College of Medicine, New York, Texas Ophthalmological Association and Section on Eye, Ear, Nose, and Throat.

Dr. Kenneth W. Warren, general surgeon, Lahey Clinic, Boston, Texas Traumatic Surgical Society.

Library Plaque Honors Benefactors

A plaque in honor of those who have made substantial contributions to the Library Endowment Fund is being placed in the Memorial Library of the Texas Medical Association this month. It has a walnut paneling base with the names of contributors on brass.

Names of those individuals and organizations appearing on the plaque are the following: Dr. and Mrs. Sam E. Thompson, Kerrville; Dr. and Mrs. E. W. Bertner, Houston; Texas Pediatric Society; Dr. Preston Hunt, Texarkana; Dr. and Mrs. N. D. Buie, Marlin; Dr. and Mrs. Judson L. Taylor, Houston; Mr. and Mrs. G. A. Ray, Pettus; past presidents and president of the Woman's Auxiliary to the Texas Medical Association; Dr. D. H. Hudgins, Forney; Dr. and Mrs. W. B. Russ, San Antonio; Dr. and Mrs. G. V. Brindley, Temple; Dr. and Mrs. V. R. Hurst, Longview; Dr. and Mrs. J. C. Terrell, Stephenville; Woman's Auxiliary to the Texas Medical Association; Dorothy Ray Lewis, Dallas; Ophthalmic Research Foundation, Austin.

Medical Students' Day Held At Baylor College of Medicine

The sixth annual Baylor University College of Medicine Medical Students' Day program was held November 21 in Houston. It was presented by the Texas Medical Association and Harris County Medical Society.

Included in the presentations were "Socio-Economic and Public Relations Considerations in Medical Practice," C. Lincoln Williston, Austin, Executive Secretary, Texas Medical Association; "Your Practice and Public Opinion," Dr. Thomas L. Royce, Houston; "Serving the Doctors of Texas," Donald M. Anderson, Assistant Executive Secretary, Texas Medical Association; "The Business Side of Your Practice," Dr. Harvey Renger, Hallettsville; "Legal Aspects of Medical Practice," Pat Bailey, Austin, Assistant General Counsel, Texas Medical Association. Dr. J. Griffin Heard, Houston, presided.

A dinner was held at the Doctor's Club in Houston, at which Dr. Donald M. Gready, Houston, presided. On the program were Daniel Cunningham, president of the senior class, and Dr. Stanley W. Olson, Houston, dean, Baylor University College of Medicine. Dinner addresses were given by Dr. R. Mayo Tenery, Waxahachie, on "Medical Ethics: Obligations of the Physician to His Colleagues and to the Public," and Dr. Howard O. Smith, Marlin, President of Texas Medical Association, on "Spectatoritis."

★ County Societies

County Society News Briefs

Dr. Walter Walthall, San Antonio, who has served a year as president-elect, will be installed as president of the *Bexar County Medical Society* at its January 14 meeting in San Antonio. Officers elected December 9 will be installed at that time also. Dr. Theordore G. Klumpp of New York, president of Winthrop Laboratories, Inc., will be the speaker for the occasion. This installation dinner is being planned as a "Friends of Medicine" night, and physicians are urged to bring their business acquaintances with them as their guests.

Officers for the *McLennan County Medical Society* who were elected at the November 11 meeting in Waco include Drs. Neill O. Simpson, president; M. W. Colgin, vice-president; J. A. Coleman, secretary; J. R. Shipp, board of censors; W. M. Avent, delegate place II; W. N. Roddy, alternate place II, all of Waco. Society members have endorsed a program to stop rheumatic fever. A committee composed of several physicians will screen several schools by classrooms in order to get an idea of the extent of the situation. Tom Wallace, director of the Hillcrest Laboratory at Waco, presented a demonstration of the artificial kidney. Dr. Howard O. Smith of Marlin, President of the Texas Medical Association, gave a talk on "The Texas State Board of Medical Examiners."

Dr. W. H. Hamrick, Houston, was chosen president-elect of the Harris County Medical Society at its November meeting in Houston. Other officers elected were Drs. William E. Sharp, Baytown, vice-president; and William H. Moorhead, Houston, secretary; and C. A. Calhoun, Houston, treasurer.

E. M. Gentry, director of narcotics for the Dallas district, spoke on the control and enforcement of narcotic regulations at a joint meeting between the members of the *Potter-Randall Counties Medical Society* and the Amarillo Pharmaceutical Association. The meeting was held November 10 in Amarillo. Seventy-three physicians and 50 pharmacists attended.

Myron I. Macht, assistant superintendent of field operations of the Cornell Automotive Injury Research Program, spoke to the *Tom Green-Eight Counties Medical Society* at its meeting November 3 in San Angelo. Mr. Macht described the different aspects of the automotive injury research program which is being subsidized by the United States Public Health Service, the Office of the Surgeon-General, the Automobile Manufacturers Association, and in Texas with the cooperation of the Texas Medical Association. Officers elected for 1959 at the meeting were Drs. Robert A. Morse, president-elect; Lloyd R. Hershberger, vice-president; Ray G. Boster, secretary-treasurer; James W. Hooker, board of censors; Sam H. Gainer, delegate; and R. F. Trotter, alternate delegate, all of San Angelo. Dr. Francis M. Spencer, also of San Angelo, will be president of the society.

Members of the *Hill County Medical Society* and the *Navarro County Medical Society* met November 11 at Hillsboro to hear C. Lincoln Williston of Austin, executive secretary of the Texas Medical Association. Mr. Williston spoke on the "Legislative Forecast, 1959."

Officers elected recently for the *Lamar-Delta Counties Medical Society* are Drs. O. R. O'Neill, president-elect; James L. Clifford, vice-president; David C. Miesch, secretary-treasurer; and N. L. Barker, delegate, all of Paris. Dr. M. A. Walker, also of Paris, is incoming president.

Dr. Odon F. von Werssowetz, medical director of the Texas Rehabilitation Center, Gonzales Warm Springs Foundation, Gonzales, spoke to the *Collin County Medical Society* at its meeting October 28 at McKinney. Dr. von Werssowetz spoke on "The Rehabilitation of Chronic Disabilities."

A panel discussion on "The Medical, Legal, and Insurance Aspects in the Treatment of Industrial Injuries" was presented as part of the program for the *Tarrant County Medical Society* at its meeting October 7 at Fort Worth. Moderator for the panel was Dr. F. C. Rehfeldt, Fort Worth. Participants in the discussion were Dr. M. P. Knight, Dallas; John A. Gooch, Fort Worth attorney; and Smith Pettigrew, medical coordinator, Texas Employers' Insurance Association, Dallas. A total of 122 members attended the meeting.

★ Woman's Auxiliary

Auxiliary's Recruitment For Paramedical Fields Proves Successful

Recruitment to encourage high school students to go into paramedical careers is one of the most interesting of many worth-while projects of the Woman's Auxiliary of the Texas Medical Association.

Although the Auxiliary has become interested in recruiting for all health fields, its initial work was in the area of nursing. In the 5 years of the existence of the Texas Association of Future Nurses in Texas, approximately 230 clubs have been organized in high schools with a

This group of future nurses pose for the Texas Medical Association photographer in a brief moment of rest during one of the Association's meetings in the headquarters building in Austin. Future nurses are often brought in during meetings to help members of the Association's staff and become better acquainted with the medical profession.

membership of more than 5,000. The association is well organized and, as of last year, completely self-supporting.

The executive board of the association includes three adult sponsors: the club sponsor of the association president, the chairman of the Committee on Careers in Nursing (representing both the Texas League for Nursing and the Texas Graduate Nurses' Association), and the chairman of Recruitment of the Woman's Auxiliary to the Texas Medical Association. This student organization has a fall executive board meeting and an annual convention, which will be held next in Galveston on March 20 and 21, 1959. The enthusiasm of the nurses-to-be is evidenced partially by the high attendance at these conventions. At the March, 1958, Fort Worth convention, the group's fourth annual meeting, more than 1,000 attended.

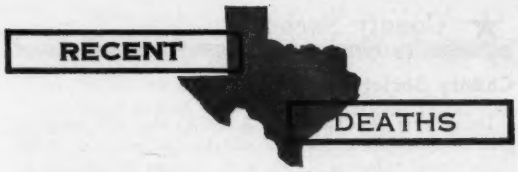
The clubs are sponsored locally by a high school sponsor (usually a teacher or school nurse) and a representative of the county medical auxiliary. With the large number of clubs, in both urban and rural locales, it is understandable that responsibilities always may not be divided equally between the two sponsors. However, it is extremely heartening to learn as often as I do, of the wonderful cooperation between the sponsors. This also helps a great deal in keeping happy medical and nursing group relations.

The clubs have instructive and active programs. The current demand for lay motion pictures from the Memorial Library is overwhelming. The clubs have learned the immeasurable value of authoritative films—now we need a far greater film supply than we presently have. Most of the clubs invite at least one interested professional speaker each month, not only for discussions of nursing, but also the allied medical fields. Field trips are taken to hospitals, clinics, health museums, public health offices and hospitals, laboratories, and other places of medical interest. It is not a case of only one or two clubs being active; many have a varied program of this type.

In many clubs the most worth-while project is that of students serving as assistants in a hospital or clinic and doing work in direct contact with nurses and doctors. This program, of course, must be well supervised and with a period of preparatory explanation before actual duty. Aside from the value of being of great service to both the patients and the hospital or clinic, the young prospective nurses have the opportunity to know and understand the patient-nurse and doctor-nurse relationships associated with the profession. Students can judge first hand whether or not they are suitable and adequate for the nursing professions which interest them. Also, some allied careers may come to their attention which they had not previously considered or even known about. All this can be discovered before the student enters the school of his choice. It is too soon to obtain statistics on this, but it seems certain to help reduce the now staggering percentages of drop-outs in our state's nursing schools.

Statistics revealed in the Texas League for Nursing's recent "Survey of Nursing Needs and Resources in Texas, 1958" suggest the progress made in the past few years by the Future Nurse Clubs. Of 20 diploma program schools reporting, 20.5 per cent of seniors had been members of future nurse clubs, whereas of the new class of freshmen, 31.4 per cent had been members, answers to the questionnaires indicate. Of the 4 degree program schools, only 7.8 per cent of the seniors were former members, whereas 30.5 per cent of the incoming freshman students had been influenced by participation in this program. It is a great increase for so short a time. It seems that the Auxiliary is helping—thanks to the enthusiasm of every interested member.

—MRS. PAUL KLINGER, San Antonio,
Chairman of Recruitment.



DR. T. WADE HEDRICK

Dr. Thomas Wade Hedrick, Abilene surgeon, died October 21, 1958, of a cerebral vascular accident (thrombosis with left hemiplegia).

A native of Wheelock in Robertson County, Dr. Hedrick was born November 27, 1890, the son of Thomas Wade and Emma (Love) Hedrick. Receiving his preliminary education at Wheelock, he also attended Southwestern University at Georgetown. He was graduated from the University of Texas School of Medicine at Galveston in 1916 and served an internship at John Sealy Hospital in Galveston. Following graduation, Dr. Hedrick practiced medicine in Robertson County for a year before serving briefly in the Army Medical Corps. He was discharged with the rank of first lieutenant in 1918 and shortly after went to Abilene, where he practiced until his death. Dr. Hedrick served for many years as chief of staff and chief surgeon at Hendrick Memorial Hospital at Abilene.

DR. T. WADE HEDRICK

The immediate past president of the Taylor County Heart Association, he was also a member of the Taylor-Jones Counties Medical Society, the Texas Medical Association, and the American Medical Association. He also belonged to the Texas Society of Gastroenterologists and Proctologists and the Southern Medical Association and was a fellow of the American College of Surgeons, being chairman of the state credentials committee for District 4. He held membership in Phi Beta Pi, the Abilene Club, and the Masonic Lodge.

Dr. Hedrick and the former Mrs. Mamie Tillett were married in Abilene in 1917. She survives as do two daughters, Mrs. John G. Russell of Dallas and Mrs. O. E. Harper of Abilene.

DR. J. L. MOET

Dr. Joe Louis Moet, La Feria, was killed in a car wreck October 20, 1958.

A native of La Feria, he was born October 23, 1915, the son of John N. and Pearl (Allen) Moet. He received his preliminary education at La Feria, attending Texas College of Arts and Industries at Kingsville and later being graduated from Baylor University College of Medicine at Dallas. His internship was served at Jefferson Davis Hospital at Houston, and after being in the United States Navy for 4 years as a lieutenant commander, he started his practice in 1946 at La Feria, continuing there until his death.

A past president of the Cameron-Willacy Counties Medical Society, he was also a member of the Texas Medical Association and the American Medical Association. Dr. Moet was the first president of the La Feria Kiwanis Club, at one time was chief of staff of Valley Baptist Hospital at Harlingen, was chairman of the board of trustees of the La Feria First Methodist Church, and was a member of the school board for the La Feria Independent School District. He enjoyed golf, hunting, fishing and had business interests in farming and citrus growing.

Dr. Moet married the former Miss Martha Fay Dicus of Harlingen. She survives as do three children, Kathryn Ann, Johnnie M., and Joe Allen Moet, and a brother, Dr. John Allen Moet of Orange Grove.

DR. J. L. MOET

DR. ROBERT BAILEY

Dr. Robert Bailey, Coleman, died July 4, 1958, of paralysis agitans.

The son of J. M. and Mary (Baines) Bailey, he was born April 28, 1882, at Harrison, Ark. Attending Coleman High School and the Agricultural and Mechanical College of Texas, he was graduated from Tulane University College of Medicine at New Orleans. Following graduation, Dr. Bailey returned to Coleman, where he practiced until 1943 when he retired because of ill health. While at Coleman he was on the staff of Overall Memorial Hospital. During World War I, Dr. Bailey served as a captain in the Medical Corps of the Army Air Corps.

A member of the Coleman County Medical Society, Dr. Bailey also belonged to the District 4 Medical Society, the Texas Medical Association, and the American Medical Association. He was elected to honorary membership in the Texas Medical Association in 1949. Dr. Bailey received two medals for Selective Service duty, and during the summer of 1958, he was given a pin for his 50 year membership in the Masonic Lodge.

On December 29, 1913, Dr. Bailey married the former Miss Nell Polk, who survives. Another survivor is his brother, Commander Claude Bailey, Burnt Hills, N. Y.

DR. WILLIAM F. KEY

Dr. William F. Key of Fort Worth died August 23, 1958, of arteriosclerotic heart disease.

Dr. Key was born February 21, 1891, in Groveton, the son of J. M. and Millie Ann (Bowman) Key. He received his preliminary education in Groveton, and attended Sam Houston State College, Huntsville, and Texas Christian University, Fort Worth. He was graduated from Baylor University College of Medicine in Dallas in 1919. He completed his internship at St. Joseph Hospital in Fort Worth and his residency at the City-County Hospital in Fort Worth. He was in general practice in Fort Worth from 1922 to 1935 when he retired because of post-encephalitis sequelae. After his retirement, he worked with the Boy Scouts 6 years at Worth Ranch during the camping season and assisted in the outdoor clinic at City County Hospital for 1 or 2 years.

Throughout his career Dr. Key was a member of the Tarrant County Medical Society, Texas Medical Association, and American Medical Association. He was made an honorary member of the state organization in 1937. He was a Master Mason, a member of the Shrine, and a member of the Christian Church. He was in the Student Army Training Corps in 1918.

Surviving Dr. Key is his wife, the former Miss Lena Mae Estill of Grapevine, whom he married August 5, 1922, in Fort Worth; two children, Dr. William Frank Key, Jr., of Clifton and Dr. Robert Estill Key of Boston, and a sister, Mrs. Ollie McClain, Groveton.

DR. BRUCE S. BARNES

Dr. Bruce Smith Barnes, Dallas, died August 17, 1958, of a heart attack while vacationing in Galveston.

Born September 24, 1920, in Evansville, Ind., Dr. Barnes was the son of Dr. and Mrs. William E. Barnes. He received a bachelor of arts degree in 1941 from DePauw University, Greencastle, Ind., and a doctor of medicine degree in 1944 from Indiana University School of Medicine, Indianapolis, ranking first scholastically in his class of 200. He interned at the Indianapolis City Hospital. After serving in the United States Army Medical Corps for 2 years, Dr. Barnes was a resident in obstetrics and gynecology at the Methodist Hospital, Dallas. He had practiced at the Chester Clinic and Hospital in Dallas since 1948, holding the position of a senior partner at the time of his death.

Dr. Barnes was a member of the Dallas County Medical Society, Texas Medical Association, and American Medical Association. He was a fellow of the American College of Obstetrics and Gynecology. He also belonged to the Lancaster Lions Club, Lancaster Presbyterian Church, Lancaster Chamber of Commerce, Oak Cliff Country Club, Oak Manor Club, Phi Gamma Delta social fraternity, Phi Beta

Pi medical fraternity, Phi Beta Kappa honorary scholastic fraternity, and Alpha Omega Alpha honorary medical fraternity. He was past president of the Indiana University Alumni Association of his area, served on the first zoning commission appointed in Lancaster, was active in Boy Scout work, and attended the Presbyterian Church. He held membership in the Planned Parenthood Association.

Survivors include his wife, Mrs. Janet B. Barnes; two sons, Bruce S. Barnes, Jr. and William Robert Barnes, both of Lancaster; his mother, Mrs. William E. Barnes, and a brother, Larry Barnes, both of Evansville, Ind.

DR. BEN H. COOLEY

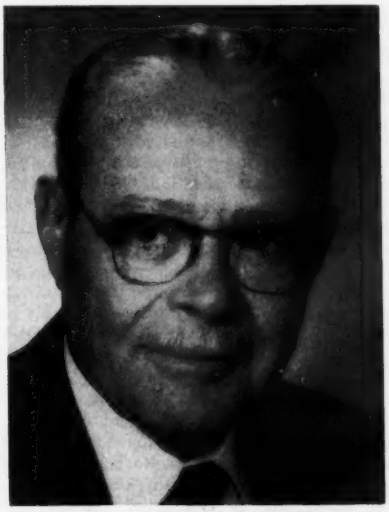
Dr. Ben Hunter Cooley, El Paso, died Stepember 22, 1958.

He was born November 26, 1894, in Smithville, and was the son of Ben Hamilton and Blanche (Wilson) Cooley. He received his preliminary education in Fort Worth public schools and was graduated from the University of Oklahoma School of Medicine in Oklahoma City in 1921. He interned at the University Hospital in Oklahoma City, later serving a 1 year residency at this same hospital.

Dr. Cooley started practice in Norman in cardiology and internal medicine in 1922, serving as instructor at the University of Oklahoma School of Medicine for 7 years. He was in the Army Medical Corps during World War I and II, working up in rank from lieutenant to colonel. He retired in 1946 to set up practice in El Paso, specializing in cardiology and internal medicine.

Former chief of staff at Southwestern General Hospital, Dr. Cooley was also chief of the medical sections at both Hotel Dieu and Providence Memorial Hospital. He was a member of the Texas Medical Association, American Medical Association, and Harris County Medical Society. He formerly had been secretary of Cleveland County Medical Society in Oklahoma for 15 years. He was a past vice-president of the Texas Heart Association, a fellow in the American College of Physicians, and a member of the Texas Academy of Internal Medicine, and was a thirty-second degree Mason. He belonged to Phi Beta Pi medical fraternity, Phi Gamma Delta social fraternity, and the Episcopal Church. His hobbies included fishing, hunting, and photography.

Survivors include his wife, the former Mrs. Eve P. Upperman of Washington, D. C., whom he married January 9, 1943, in Silver City, N. Mex.; three daughters, Mrs. Robert H. Palm, El Paso; Mrs. Taylor Green, Houston; and Mrs. Earl Smith, Denver; and one sister, Mrs. Floyd Lee, Norman.



DR. BEN H. COOLEY

DR. ELLA WARE

Dr. Ella Ware, formerly of Stockdale, died October 29, 1958, at San Antonio of cerebral hemorrhages.

Dr. Ware was born May 13, 1870, at Riddleville (now Gillette) in Karnes County and was reared near Stockdale. Attending public schools at Stockdale, she later became the first woman to be graduated under the 4 year plan from the University of Texas Medical Branch at Galveston, receiving her degree in 1899. Rejecting the offer of a professorship to enter general practice, Dr. Ware practiced in the Stockdale area, doing work not only in Wilson County, but also in Karnes, Gonzales, Guadalupe, and Bexar Counties. She took time out periodically for refresher courses and other postgraduate work in New York, Chicago, and Galveston. After 50 years of practicing, Dr. Ware retired because of ill health, later moving to San Antonio, where she stayed until her death. In 1952 Dr. Ware was given a certificate of appreciation by the Galveston Medical Branch. In 1954, she was honored at "Ella Ware Day" in Stockdale.

DR. ELLA WARE

A past president of the Karnes-Wilson Counties Medical Society, Dr. Ware was elected to honorary membership in the Texas Medical Association in 1958. Dr. Ware was a member of the First Baptist Church at Stockdale and a member of the Stockdale Chapter, Order of Eastern Star.

Members of the family who survive include seven sisters, Mrs. L. E. Seely, Mrs. William H. King, and Miss Mayme Ware, all of San Antonio; Mrs. Alma W. Crosby and Mrs. C. D. Bulger, Beaumont; Mrs. A. L. Brown and Miss Lois Ware, Del Rio; and one brother, J. R. Ware, Stockdale.

DOES NOT CIRCULATE

DECEMBER, 1958

TEXAS State Journal of Medicine

VOLUME 54 • NUMBER 12 • AUSTIN, TEXAS

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APRIL 18-21
SAN ANTONIO

Diabetes

Incisional Hernia

County Officials Conference, Legislative Symposium, Orientation

Annual Session Guests

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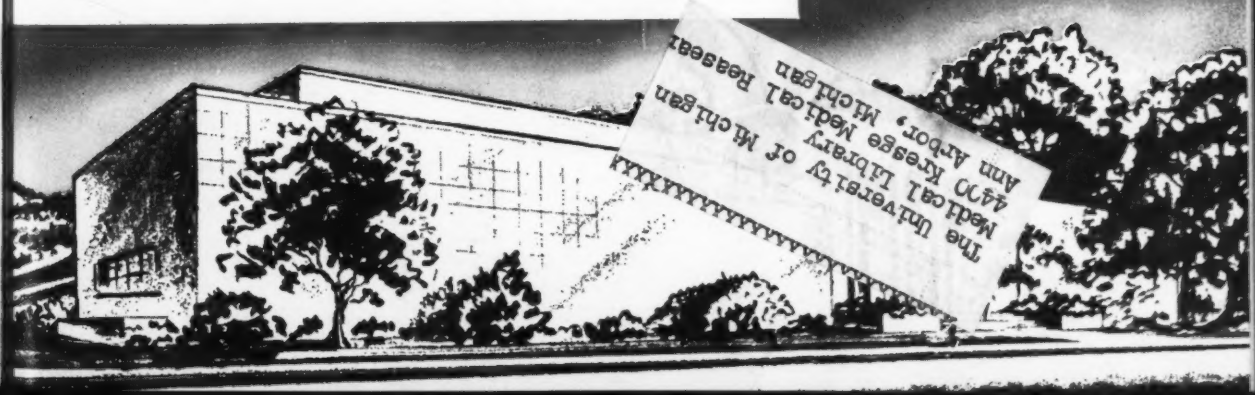

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(1) Hagedorn, A. B.: Proc. Staff Meet. Mayo Clin. 32:705 (Dec. 11) 1957.
(2) Best, W. R.; Louis, J., and Limarzi, L. R.: M. Clin. North America (Jan.) 1958, p. 3.

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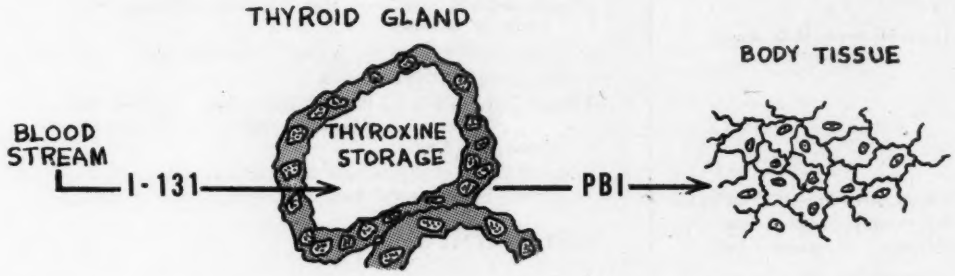
THE THYROID PROFILE

1. WHAT ARE THE ACCEPTED TESTS FOR DETERMINING THYROID FUNCTION?

Tests available are the Protein-Bound-Iodine, the I-131 Uptake, the Blood Cholesterol and the BMR.

2. WHAT FUNCTION OF THE THYROID IS SHOWN BY THE I-131 AS COMPARED TO THE PBI?

The following diagram shows the points in thyroid function that are studied.



The I-131 shows the amount of iodine that the thyroid needs for its production of thyroxine, and the PBI measures the finished product.

3. HOW IS THE BMR AND THE CHOLESTEROL LEVEL RELATED?

The results are usually reciprocally related. If the metabolic rate is low, the cholesterol level is elevated, and vice versa.

4. WHAT INTERFERES WITH THE VARIOUS TESTS?

Iodine administration by mouth or parenterally for X-ray studies will interfere with the I-131 Uptake and the PBI. The BMR is the least accurate of the procedures; mechanical difficulties with the equipment, poor preparation of the patient, anxiety, etc., will interfere with this test.

5. WHAT TESTS ARE PREFERRED?

The PBI and the I-131 Uptake. Both are good tests for the diagnosis of thyroid malfunction. The PBI and the BMR should usually be used to follow a patient under treatment.

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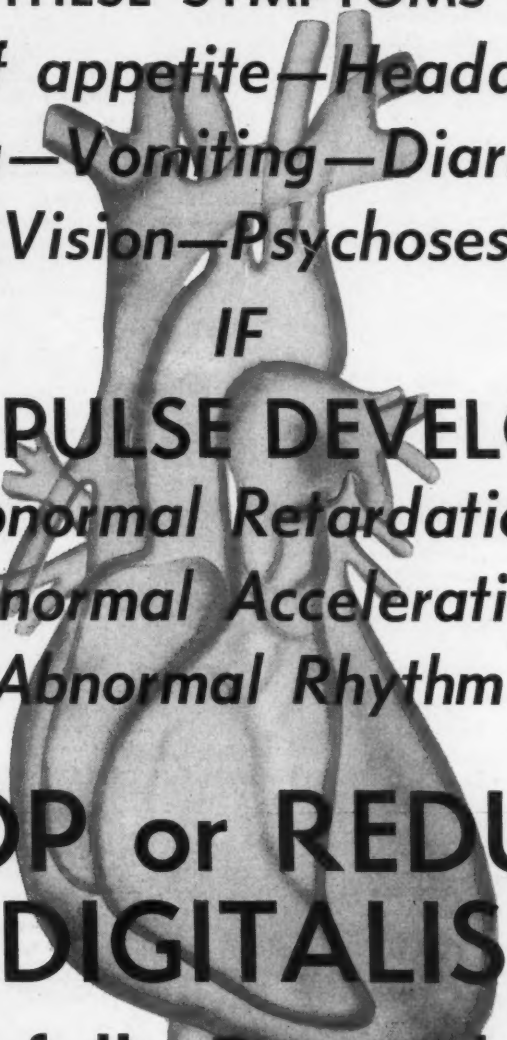

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References:

1. Boland, E.W.: California Med. 88:417 (June) 1958.
2. Bunim, J.J., et al.: Arthr. & Rheum. 1:313 (Aug.) 1958.
3. Boland, E.W., and Headley, N.E.: Paper read before the Am. Rheum. Assoc., June 21, 1958, San Francisco, Cal.
4. Bunim, J.J., et al.: Paper read before the Am. Rheum. Assoc., June 21, 1958, San Francisco, Cal.

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With proper adjustment of dosage, treatment may ordinarily be changed over to DECADRON from any other corticosteroid on the basis of the following milligram equivalence:

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↓ One 4 mg.	↓ One 5 mg.	↓ One 20 mg.	↓ One 25 mg.
tablet of methylprednisolone or triamcinolone	tablet of prednisolone or prednisone	tablet of hydrocortisone	tablet of cortisone

SUPPLIED:

As 0.75 mg. scored pentagon-shaped tablets; also as 0.5 mg. tablets to provide maximal individualized flexibility of dosage adjustment.

Detailed literature is available to physicians on request.

*DECADRON is a trademark of Merck & Co., Inc.
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Decadron*

DEXAMETHASONE

ANNOUNCING

a new order of magnitude in corticosteroid therapy!

The great corticosteroid era
opened ten years ago
with the introduction of CORTONE® (cortisone).
Today, MERCK SHARP & DOHME proudly
presents the crowning
achievement of the first corticosteroid
decade—DECADRON (dexamethasone)
—a new and unique compound, which
brings a new order of magnitude
to corticosteroid therapy

Decadron*

DEXAMETHASONE

to treat more patients more effectively

MSD MERCK SHARP & DOHME

our phone is never out of reach!

Sound like a tall Texas tale? Well we'll bet a Stetson we can fill your needs faster than you thought was possible. Give us a try and see for yourself.

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In emergencies, just call us: Fort Worth, WA 6-4649

FIFTH AVE. AND MAGNOLIA · P. O. BOX 310 · FORT WORTH, TEXAS

TS-04

Now—All cold symptoms can be controlled

This new timed-release tablet provides:

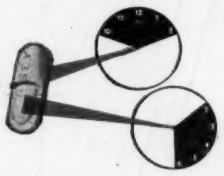
- ***...the superior decongestant and antihistaminic action of Triaminic***
- ***...non-narcotic cough control as effective as with codeine, but without codeine's drawbacks***
- ***...an expectorant to help the patient expel thickened mucus***
- ***...the specific antipyretic and analgesic effect of well-tolerated APAP***
- ***...the prompt and prolonged activity of timed-release medication***

Each* TUSSAGESIC *Tablet contains:

TRIAMINIC®	50 mg.
(phenylpropanolamine HCl	25 mg.;
pheniramine maleate	12.5 mg.;
pyrilamine maleate	12.5 mg.)
Dormethan (brand of dextromethorphan HBr)	30 mg.
Terpin hydrate	180 mg.
APAP (N-acetyl-p-aminophenol)	325 mg.

Also available:
for those who prefer liquid medication—
Tussagesic suspension
In each 5 ml.: Triaminic, 25 mg.; Dormethan, 15 mg.; terpin hydrate, 90 mg.; APAP, 120 mg.

Tussagesic timed-release tablets provide relief in minutes, which lasts for hours



first—3 to 4 hours of relief from the outer layer

then—3 to 4 more hours of relief from the inner core

Dosage: 1 tablet in the morning, mid-afternoon, and evening, if needed. Should be swallowed whole to preserve the timed-release action.
Suspension: Adults—1-2 tsp. every 3-4 hours; Children 6-12 years old—1 tsp. every 3-4 hours; Children under 6—dosage in proportion.

NEW Tussagesic*

*Contains TRIAMINIC to STOP running noses and open stuffed noses orally

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska • Peterborough, Canada

If you were to examine these patients

could you detect the asthmatic on Medrol*?

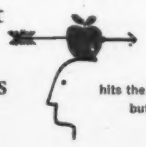
Probably not. Not without a history.

First, because he's more than likely symptom-free.

Second, because he shows none of the disturbing changes in appearance, behavior or metabolism sometimes associated with corticotherapy.

Even your practiced clinical eye would find it difficult to spot someone else's Medrol patient.

But in your own patients, you could see the advantages of Medrol right away. Why not try it?



Medrol hits the **disease,** but spares the **patient**

Upjohn

The Upjohn Company, Kalamazoo, Michigan

*TRADEMARK, REG. U. S. PAT. OFF.—METHYLPREDNISOLONE, UPJOHN

Give your diagnostic skill the advantage of **MODERN** instrumentation

PRICES DELIVERED
CONTINENTAL U. S. A.

Whenever a diagnostic "tool" can give you some *added* advantage in *better* performance or *wider* usefulness — your own diagnostic skill is aided by *more complete facts*, and your time is saved through simpler, more convenient use. *Each of these Sanborn instruments gives you just such added advantages.*

With the new Rappaport-Sprague Acoustic Stethoscope, sounds which are only faintly discernible or at the threshold of audibility with conventional stethoscopes become clearly audible, providing new assurance in auscultation. Equipped with five chest pieces for sensing and localizing sounds of various pitch, and three sets of ear pieces for proper fit, this new Stethoscope clearly reflects the results of ten years of research and investigation undertaken during its development.

In the Visette electrocardiograph, *true* portability in a clinically accurate ECG is now a practical reality. By its brief case size and 18-pound weight, the Visette lets you take 'cardiography *to your patient* — in his home, at the hospital, at an industrial plant clinic, wherever the need exists. Modern electronic components — a new, much lighter galvanometer — design innovations ranging from pushbutton grounding and double-check standardization signals to fully automatic stylus stabilization as leads are switched — make the Visette the most *convenient* ECG you (and your technician) can use. And this first (and still the only) 18-pound 'cardiograph is now *being used* by more than 3000 doctors, both here and abroad.

For the benefits modern instrumentation can give you and your patients — by extending your diagnostic abilities and saving your time in day-to-day practice — ask your local Sanborn man for complete facts on these two unusual instruments. He will also be glad to tell you how you may use a Visette for 15 days in your own practice *without cost or obligation*, through the exclusive Sanborn "Try-Before-Buying" plan. Call or write him soon — or address Inquiry Director at the main office in Waltham, Mass.

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*Compazine**

nausea and vomiting

—from virtually any cause

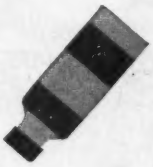
- in pregnancy—pre- and postoperative states—gastroenteritis—alcoholism—cancer and chronic diseases
- control is achieved with low dosage—usually 15 to 20 mg. daily—and often within a half hour after the first oral dose

'Compazine' is remarkable for its freedom from drowsiness. Patients carry on normal activities and often experience an actual alerting effect.

...for immediate control of severe vomiting:

Ampuls, 2 cc. (5 mg./cc.)

NEW: Multiple dose vials, 10 cc. (5 mg./cc.)



—always carry one in your bag

Also available:

Tablets, 5, 10 and 25 mg., in bottles of 50 and 500.

Spansule† *capsules*, 10, 15 and 30 mg., in bottles of 30 and 250.

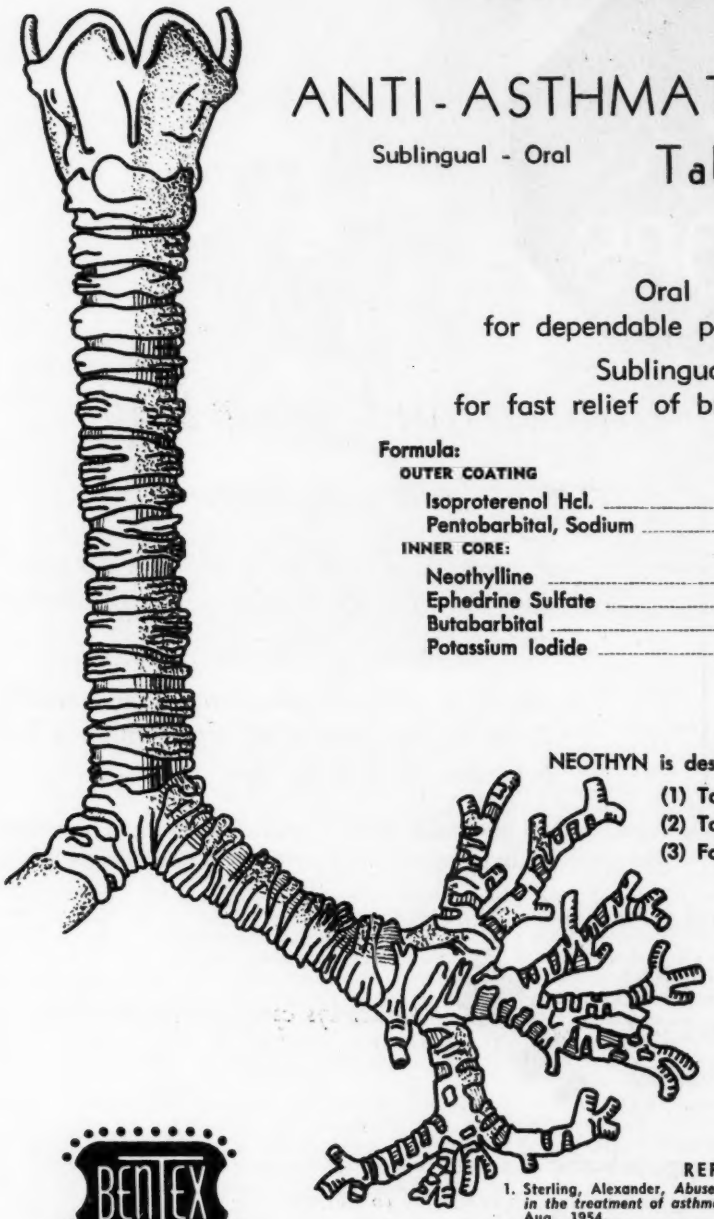
Suppositories, 5 and 25 mg., in boxes of 6.

Syrup, 5 mg./teaspoonful (5 cc.), in 4 fl. oz. lightproof bottles.

Smith Kline & French Laboratories, Philadelphia

*T.M. Reg. U.S. Pat. Off. for prochlorperazine, S.K.F.
†T.M. Reg. U.S. Pat. Off. for sustained release capsules, S.K.F.

NEOTHYN



ANTI-ASTHMATIC

Sublingual - Oral **Tablet**

Oral
for dependable prophylaxis
Sublingual
for fast relief of bronchospasm

Formula:

OUTER COATING

Isoproterenol Hcl.	5 mg.
Pentobarbital, Sodium	1/6 gr.

INNER CORE:

Neothylline	2½ gr.
Ephedrine Sulfate	⅜ gr.
Butabarbital	1/6 gr.
Potassium Iodide	5 gr.

NEOTHYN is designed—

(1) To treat the acute attack;
(2) To prevent an impending attack;
(3) For daily prophylactic use.

PARTICULARLY EFFECTIVE
in
LIQUEFACTION
of
TENACIOUS MUCOUS

BENTEX PHARMACEUTICAL CO.

3408 CAROLINE STREET
HOUSTON 4, TEXAS

REFERENCES:

1. Sterling, Alexander, *Abuse of epinephrine and related compounds in the treatment of asthmatics*, Am. Pract. & Digest Treat. 5:595, Aug., 1954.
2. Siegmund, O. A., Beglin, Nettie, and Lands, A. M., *The bronchodilator activity of some analogs of N-isopropylarterenol (Isoproterenol)*, J. Pharmacol. & Therap. 97:14, Sept., 1949.
3. Dr. Hoobler, Chief of Cardio Vascular diseases, University of Michigan Hospital, *Treatment of Asthma*, December 22, 1954.
4. *U. S. Dispensatory, Publisher:* J. P. Lippincott Company.

SAMPLES AND LITERATURE TO PHYSICIANS ON REQUEST.

new

Tao*

PRONOUNCED TAY-O

Capsules / Oral Suspension

designed for effective control of common gram-positive infections

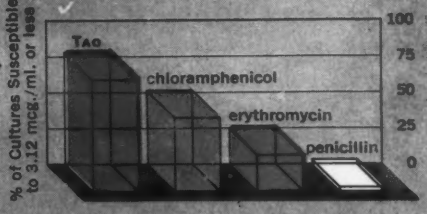
effective . . .

CLINICAL RESULTS	adults	children	all Staph infections
Cured	172 (80%)	148 (89%)	71 (88%)
Improved	28 (13%)	8 (5%)	7 (9%)
Failure	17 (7%)	11 (6%)	3 (3%)

Types of infecting organisms: The majority of identified etiologic microorganisms were Staph. aureus and Staph. albus. TAO has its greatest usefulness against organisms such as: staphylococci (including strains resistant to other antibiotics), streptococci (beta-hemolytic strains, alpha-hemolytic strains and enterococci), pneumococci, gonococci, Hemophilus influenzae.

even against resistant staph[1,2] . . .

Per cent of "antibiotic-resistant" epidemic staphylococci cultures susceptible to TAO, erythromycin, penicillin and chloramphenicol.[1]



well tolerated . . .

REACTIONS:

(a) adults	(b) children
Total—9.2% (20 out of 217)	Total—0.6% (1 out of 167)
Skin rash—1.4% (3 out of 217)	Skin rash—none
Gastrointestinal—7.8% (17 out of 217)	Gastrointestinal—0.6% (1 out of 167)

There was complete freedom from adverse reactions in 94.5% of all patients. Side effects in the other 5.5% were usually mild and seldom required discontinuance of therapy.

. . . stability in gastric acid • rapid, high and sustained blood levels • high urinary concentrations • outstanding palatability in a liquid preparation.

Dosage and Administration: Dosage varies according to the severity of the infection. For adults, the average dose is 250 mg. q.i.d.; to 500 mg. q.i.d. in more severe infections. For children 8 months to 8 years of age, a daily dose of approximately 30 mg./Kg. body weight in divided doses has been found effective. Since TAO is therapeutically stable in gastric acid, it may be administered at any time, without regard to meals.

Supplied: TAO Capsules—250 mg. and 125 mg.; bottles of 60. TAO for Oral Suspension—1.5 Gm.; 125 mg. per teaspoonful (5 cc.) when reconstituted; unusually palatable cherry flavor; 2 oz. bottle.

References: 1. English, A. R., and Fink, F. C.: Antibiotics & Chemother. (Aug.) 1958. 2. English, A. R., and McBride, T. J.: Antibiotics & Chemother. (Aug.) 1958. 3. Wennersten, J. R.: Antibiotic Med. & Clin. Therapy (Aug.) 1958. 4. Celmer, W. D., et al.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia, Inc., 1958, p. 476.

NEW YORK 17, N. Y.

Chas. Pfizer & Co., Inc.

SCIENCE FOR THE WORLD'S WELL-BEING

TRADEMARK

AN AMES CLINIQUICK

CLINICAL BRIEFS FOR MODERN PRACTICE

if your patient wears tinted glasses and sighs frequently...?

She may have an anxiety state. The tinted glasses may be worn as a shield against the world—and to relieve the photophobia resulting from pupillary dilatation caused by anxiety-induced hyperadrenalism. The sighs may be a result of fatigue from emotional unrest.

Source—Meyer, O. O.: Northwest Med. *53*:1006, 1954.

*4 findings from a recent study**

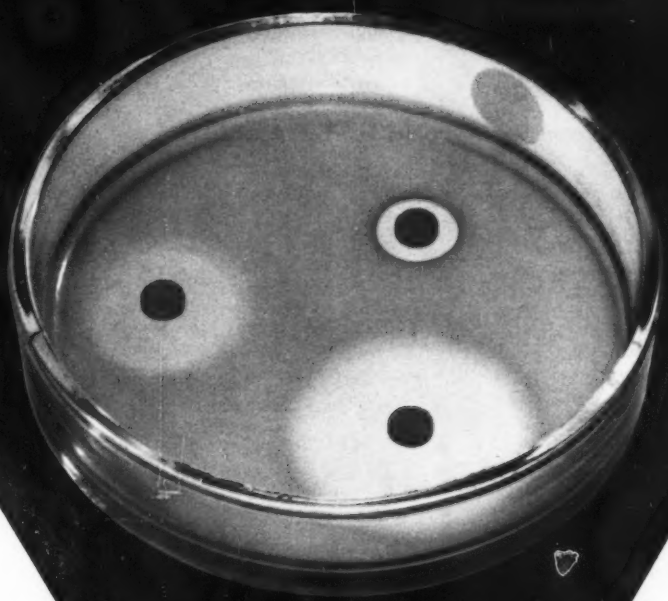
calmative **nostyn**® Ectylurea, AMES (2-ethyl-*cis*-crotonylurea)

1. Anxiety and nervous tension appeared to be most benefited by NOSTYN.
2. Seventy per cent of patients obtained some degree of relief.
3. Greater inward security and serenity were experienced and expressed.
4. Mental depression did not develop in patients previously depressed by meprobamate or a similar drug.

dosage: 150-300 mg. (½ or 1 tablet) three or four times daily. *supplied:* NOSTYN tablets, 300 mg., scored. Bottles of 48 and 500.

*Bauer, H. G.; Seegers, W.; Krawzoff, M., and McGavack, T. H.: New York J. Med. *58*:520 (Feb. 15) 1958.

A



for
the control
of all
coccal
infections

abbott's antibiotic triad

Erythrocin®

stearate

(Erythromycin Stearate, Abbott)

indications:

In infections caused by staphylococci, streptococci (including enterococci) and pneumococci. Also, against organisms that have become resistant to other antibiotics. ERYTHROCIN should be used where patients are allergic to penicillin or other antibacterials.

dosage:

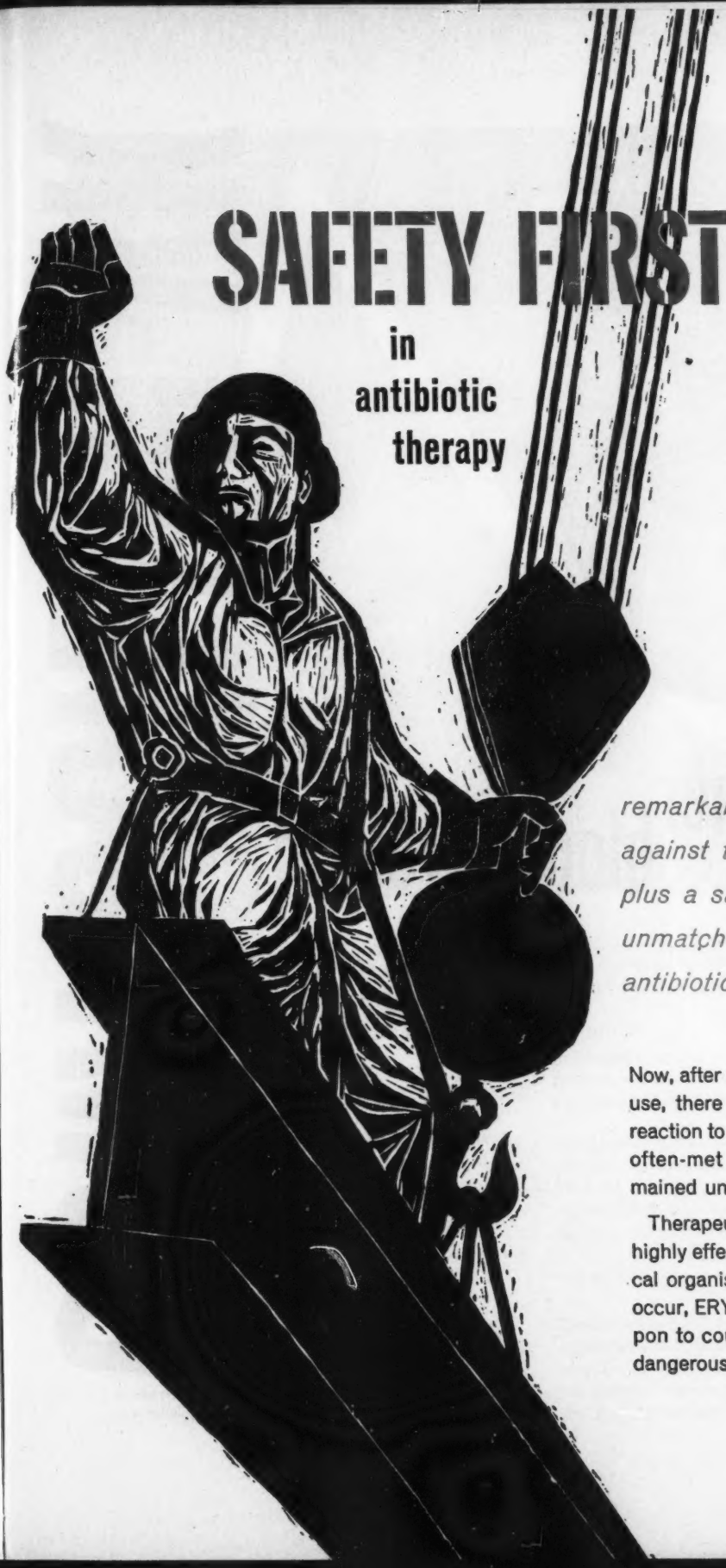
Usual adult dose is 250 mg. every six hours; for severe infections, usual dose is 500 mg. every six hours. Child's dose may be reduced in proportion to body weight.

supplied:

In bottles of 25 and 100 Filmtabs (representing 100 and 250 mg. of ERYTHROCIN activity). Also, in cinnamon-flavored oral suspension; 75-cc. bottles. Each 5-cc. teaspoonful represents 100 mg. of ERYTHROCIN activity.

® Filmtab—Film-sealed tablets, Abbott; pat. applied for.

© 1958, ABBOTT LABORATORIES, NORTH CHICAGO, ILLINOIS



SAFETY FIRST

in antibiotic therapy

remarkable effectiveness against the cocci– plus a safety record unmatched in systemic antibiotic therapy

Now, after more than six years of extensive use, there has not been a single serious reaction to ERYTHROCIN. Additionally, the often-met problem of resistance has remained unusually low with ERYTHROCIN.

Therapeutically, you'll find ERYTHROCIN highly effective against the majority of coccal organisms. Where severe viral attacks occur, ERYTHROCIN may well be the weapon to counteract those dangerous complications.

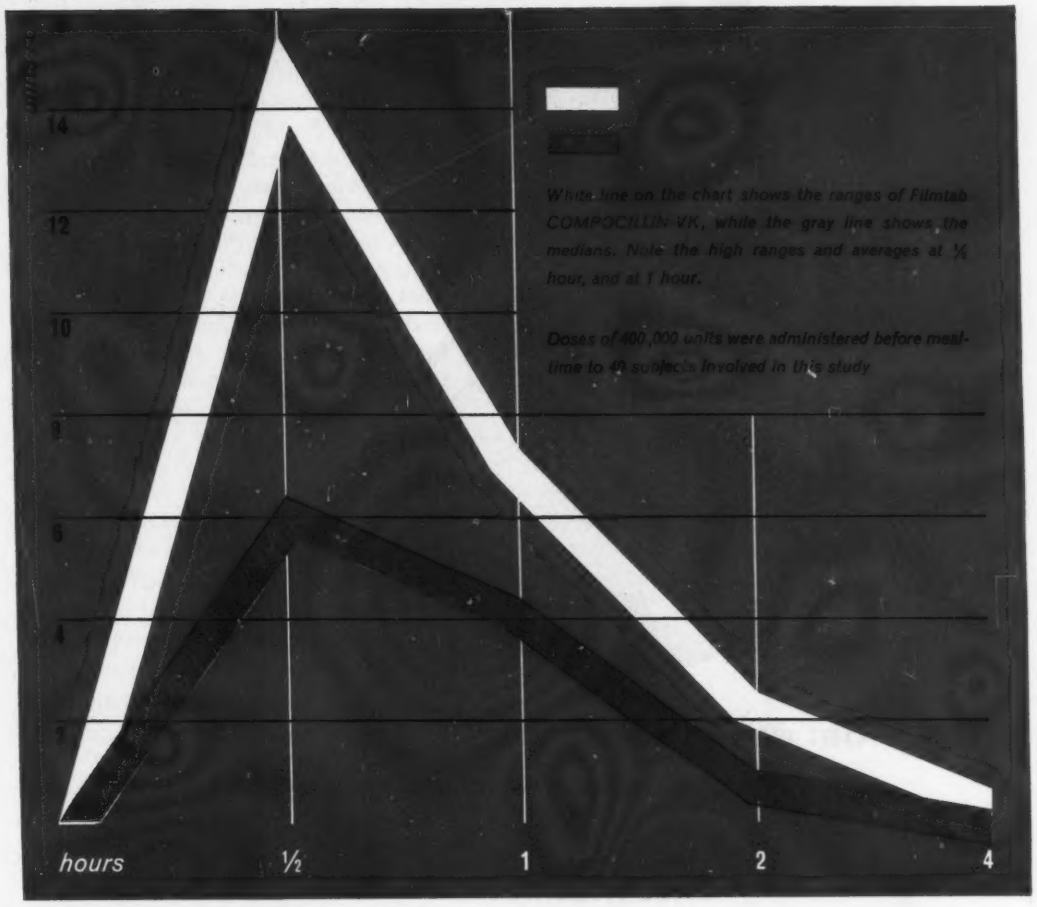
Abbott

COMPOCILLIN-VK®

* Potassium
Penicillin V

the higher blood levels of

809136


14
12
10
8
6
4
2
hours
½
1
2
4
White line on the chart shows the ranges of Filmtab COMPOCILLIN-VK, while the gray line shows the medians. Note the high ranges and averages at ¼ hour, and at 1 hour.
Doses of 400,000 units were administered before meal-time to 40 subjects involved in this study

NOW, IN BOTH FILMTAB AND ORAL SOLUTION, patients get high penicillin V blood levels with COMPOCILLIN-VK. Note the chart. Concentrations are three times higher than an equivalent dose of potassium penicillin G.

COMPOCILLIN-VK is indicated whenever you desire oral penicillin therapy. In severe infections, oral penicillin should be supplemented by parenteral therapy to obtain the maximum therapeutic response.

Indications:

Against all organisms sensitive to oral penicillin therapy. For prophylaxis and treatment of complications in viral conditions. And as a prophylaxis in rheumatic fever and rheumatic heart disease.

Dosage:

Depending on the severity of the infection, the usual adult dose is 125 to 250 mg. (200,000 to 400,000 units) every four to six hours. For children, dosage may be reduced in proportion to body weight.

Supplied:

In Filmtabs, representing 125 mg. (200,000 units) of potassium penicillin V, bottles of 50 and 100. In 250 mg. (400,000 units), bottles of 25 and 100.

For Oral Solution, COMPOCILLIN-VK comes in dry granules for easy reconstitution with water. Cherry-flavored, the granules come in 40-cc. and 80-cc. bottles. Each 5-cc. teaspoon of solution represents 125 mg. (200,000 units) of potassium penicillin V.

COMPOCILLIN-V® Oral Suspension (Ready-Mixed), Hydrabamine Penicillin V, Abbott, comes in 40-cc. and 80-cc. bottles. Each tasty, banana-flavored 5-cc. teaspoonful represents 180 mg. (300,000 units) of penicillin V. At all pharmacies. Abbott

the most effective antibiotic available against staphylococci

CRYSTALLIZED

SPONTIN®

(RISTOCETIN, ABBOTT)

PREPARED FROM PURE CRYSTALS

Provides Outstanding Clinical Effectiveness Against Coccal Infections, Including Resistant Staphylococci and Enterococci[1]

Provides Bactericidal Action Against Coccal Infections[1]

Provides Successful Short-Term Therapy In Endocarditis[2]

612180

Now, after just 12 months, SPONTIN has become an outstanding drug of choice against resistant staphylococci, and in other serious coccal infections.

Six papers presented at the Antibiotics Symposium[1] reported the effectiveness of SPONTIN against resistant staphylococcal infections. Clinical reponses involved enterococcal endocarditis, staphylococcal pneumonias and staphylococcal bacteremias. Many of these patients were going downhill steadily—in spite of treatment by other antibiotics.

Toxicity? Careful attention to dosage recommendations has practically eliminated toxicity and side effects as serious obstacles to therapy. Also, recent improvements have been made in the manufacture of SPONTIN; the drug is now made from pure crystals. A recent report[3] in the Journal of the American Medical Association concluded, "It is our opinion that, if proper precautions are observed, ristocetin is a safe and potent agent to employ in the treatment of staphylococcal infections."

If you do not have the revised literature on this lifesaving antibiotic, please contact your Abbott Representative soon; or write direct to Abbott Laboratories, North Chicago, Illinois.

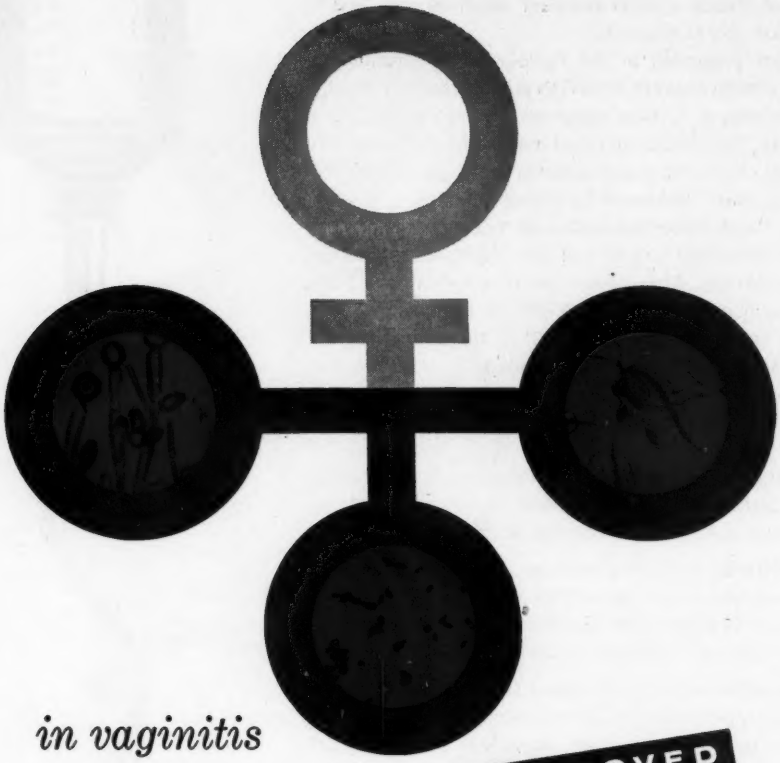
INDICATIONS: Against a wide range of staphylococcal, streptococcal, pneumococcal and enterococcal infections. A drug of choice for treating serious infections, particularly those caused by organisms that resist all other antibiotics.

DOSAGE: Administered intravenously. In pneumococcal, streptococcal and enterococcal infections, a dosage of 25 mg./Kg. will usually be adequate. Majority of staphylococcal infections will be controlled by 25 to 50 mg./Kg. per day. It is recommended that the daily dosages be divided into two or three equal parts at eight- or 12-hour intervals.

SUPPLIED: In vials containing a sterile, lyophilized powder, representing 500 mg. of ristocetin A activity.
Be sure your hospital has it stocked.

Abbott

1. Sixth Annual Symposium on Antibiotics, Washington, D. C., Oct. 15, 16, 17, 1958.
2. Antibiotics Annual, 1957-58, p. 187-98.
3. J.A.M.A., 167:1584, July 26, 1958.



in vaginitis

IMPROVED

TRICOFURON®

destroys all 3 *principal pathogens*

Whether vaginitis is caused by Trichomonas, Monilia or Hemophilus vaginalis—alone or combined—TRICOFURON IMPROVED swiftly relieves symptoms and malodor, and achieves a truly high percentage of cultural cures, frequently in 1 menstrual cycle. TRICOFURON IMPROVED provides: a *new* specific moniliacide MICOFUR® brand of nifuroxime, the *established* specific trichomonacide FUROXONE® brand of furazolidone and the *combined* actions of both against Hemophilus vaginalis.

1. *Office* insufflation once weekly of the Powder (MICOFUR [*anti*-5-nitro-2-furaldoxime] 0.5% and FUROXONE 0.1% in an acidic water-soluble powder base). **2.** Continued *home* use twice daily, with the Suppositories (MICOFUR 0.375% and FUROXONE 0.25% in a water-miscible base).

℞ NEW BOX OF 24 SUPPOSITORIES WITH APPLICATOR FOR MORE PRACTICAL AND ECONOMICAL THERAPY.

NITROFURANS—a *new* class of antimicrobials—neither antibiotics nor sulfonamides.

EATON LABORATORIES, NORWICH, NEW YORK

for fever and pain / in infants and children

when your little patient

is burning with fever.....

Tempra®

Acetaminophen, Mead Johnson

syrup • drops

brings relief quickly...conveniently

- ___ safe
- ___ well tolerated
- ___ easy to give
- ___ tastes good

Mead Johnson

Symbol of service in medicine

TE403

IN OFFICE SURGERY

ELECTIVE AND TRAUMATIC

use XYLOCAINE *first...*
as a local anesthetic
or a topical anesthetic

SWAB **SPRAY** **INFILTRATION** **NERVE BLOCK**

Xylocaine HCl solution, the versatile anesthetic for general office surgery, relieves pain promptly and effectively with adequate duration of anesthesia. It is safe and predictable. Local tissue reactions and systemic side effects are rare. Supplied in 20 cc. and 50 cc. vials; 0.5%, 1% and 2% without epinephrine and with epinephrine 1:100,000; also in 2 cc. ampules; 2% without epinephrine and with epinephrine 1:100,000.

XYLOCAINE® HCl SOLUTION
(brand of lidocaine*)

Astra Pharmaceutical Products, Inc., Worcester 6, Mass., U.S.A.

SEE PDR

*U.S. PAT. NO. 2,441,498 MADE IN U.S.A.

Proven

in over three years of clinical use
in over 600 clinical studies

Specific

FOR RELIEF OF ANXIETY
AND MUSCLE TENSION

Selective

Does not interfere with autonomic function
Does not impair mental efficiency,
motor control, or normal behavior
Has not produced hypotension,
agranulocytosis or jaundice

MEPROBAMATE (WALLACE)

Miltown®

Supplied: 400 mg. scored tablets, 200 mg. sugar-coated tablets.

WALLACE LABORATORIES, New Brunswick, N. J.

CH-8049

nasal and paranasal congestion and control secondary invaders

Now, a single unique preparation, Trisulfaminic, can provide dramatic relief from congestion, and at the same time protect the patient from secondary bacterial invaders. Often within minutes of the first dose, congestion begins to clear; the patient can breathe again.

Trisulfaminic is particularly valuable for the "almost well" patient who is recovering from influenza but is left with congested nasal and bronchial passages. And for patients with purulent rhinitis, sinusitis or tonsillitis, combination therapy with Trisulfaminic offers a most realistic approach to total treatment.

Oral Decongestant Action. Through the action of Triaminic, nasal patency is achieved rapidly and dramatically. Adequate ventilation helps eliminate mucus-harbored pathogens. And because Trisulfaminic is administered *orally,* there is no problem of rebound congestion, no pathological change wrought in the nasal mucosa.

Wide-Spectrum Action. Secondary bacterial infections, which are always a threat in upper respiratory involvement, are forestalled by the wide-spectrum effectiveness of triple sulfonamides. This added antibacterial protection makes Trisulfaminic highly useful in treating the debilitated patient who is prone to lingering or frequently recurring colds.

Trisulfaminic *tablets and suspension*

TRIAMINIC PLUS TRIPLE SULFAS

Each Tablet and each 5 ml. teaspoonful of Suspension contains:

Triaminic®	25 mg.
(phenylpropanolamine HCl	12.5 mg.;
pheniramine maleate	6.25 mg.;
pyrilamine maleate	6.25 mg.)
Trisulfapyrimidines U.S.P.	0.5 Gm.

Dosage: Adults—2 to 4 tablets or teaspoonfuls initially, followed by 2 tablets or teaspoonfuls every 4 to 6 hours until the patient has been afebrile for 3 days. Children 8 to 12 years—2 tablets or teaspoonfuls initially, followed by 1 tablet or teaspoonful every 6 hours. Younger children—dosage in proportion.

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska • Peterborough, Canada

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

INTENSIVE POSTGRADUATE COURSES STARTING DATES—EARLY 1959

SURGERY—Surgical Technic, Two Weeks, February 2, February 16.
Surgery of the Colon & Rectum, One Week, March 2, April 6.
Fractures & Traumatic Surgery, Two Weeks, March 9.
Treatment of Varicose Veins, Two Days, February 2, March 2.
American Board Review Course, Two Weeks, April 6.
Blood Vessel Surgery, One Week, March 2.
Gallbladder Surgery, Three Days, March 30.
Surgery of Hernia, Three Days, April 2.

GYNECOLOGY & OBSTETRICS—
Office & Operative Gynecology, Two Weeks, February 9, March 16.
Vaginal Approach to Pelvic Surgery, One Week, February 2, March 9.
General & Surgical Obstetrics, Two Weeks, February 23, March 30.

MEDICINE—Electrocardiography, Two-Week Basic Course, March 16.
Gastroscopy & Gastroenterology, Two Weeks, March 2.
American Board Review Course, One Week, April 20.

UROLOGY—Two-Week Intensive Course, March 30.
Ten-Day Practical Course in Cystoscopy, by appointment.

RADIOLOGY—Diagnostic X-Ray, Two Weeks, March 2, April 27.
Clinical Uses of Radioisotopes, Two Weeks, May 4.

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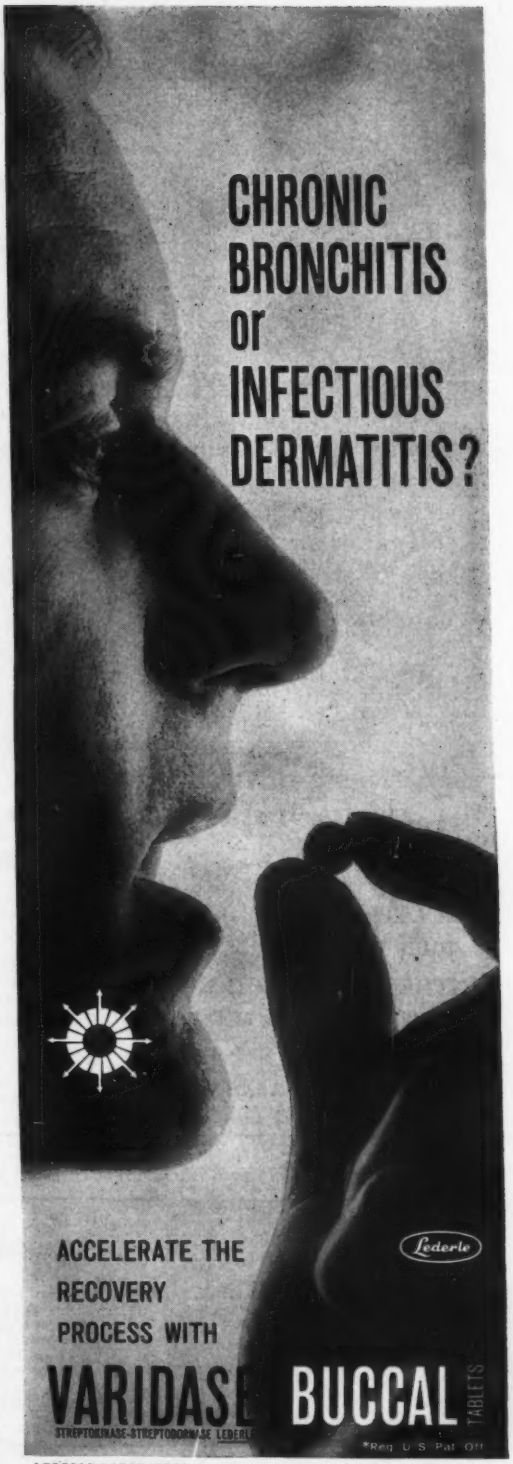

LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Of course, *women like "Premarin"*

Therapy for the menopause syndrome should relieve not only the psychic instability attendant the condition, but the vasomotor instability of estrogen decline as well. Though they would have a hard time explaining it in such medical terms, this is the reason women like "Premarin."

Doctors, too, like "Premarin," because it really relieves the symptoms of the menopause. It doesn't just mask them – it replaces what the patient lacks – natural estrogen.

"PREMARIN"®
conjugated estrogens (equine)

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IN URTICARIA AND PRURITUS

VISTARIL*

HYDROXYZINE PAMOATE

A PSYCHOTHERAPEUTIC ANTIHISTAMINE

(as designated by A.M.A. Council on Drugs, 1958)

SPECIFIC ANTIHISTAMINIC ACTION in the treatment of a variety of skin disorders commonly seen in your practice.

"While some of the tranquilizers are only partially effective as far as antiallergic activities are concerned . . . [hydroxyzine] has been found, by comparison, to be the most potent thus far . . ."[1]

"The most striking results were seen in those patients with chronic urticaria of undetermined etiology."[2]

PLUS

PSYCHOTHERAPEUTIC POTENCY for the relief of anxiety and tension.

The psychotherapeutic effectiveness of hydroxyzine (VISTARIL) was confirmed in a series of 479 patients suffering from a wide variety of dermatoses, including atopic dermatitis, neurodermatitis, psoriasis, lichen planus, nummular eczema, dyshidrosis, pruritus ani and vulvae, and rosacea. "Adverse reactions were minimal."[3]

RECOMMENDED ORAL DOSAGE: *50 mg. q.i.d. initially; adjust according to individual response.*

VISTARIL Capsules: *25 mg., 50 mg., 100 mg.*

VISTARIL Parenteral Solution: *10 cc. vials and 2 cc. Steraject® Cartridges. Each cc. contains 25 mg. hydroxyzine (as the HCl).*

More than a tranquilizer

REFERENCES:

1. Eisenberg, B. C.: Clinical Medicine *5*:897-904 (July) 1958.
2. Feinberg, A. R., et al.: J. Allergy *29*:358 (July) 1958.
3. Robinson, H. M., et al.: So. Med. J. *50*:1282 (Oct.) 1957.

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PFIZER LABORATORIES *Division, Chas. Pfizer & Co., Inc., Brooklyn 6, N. Y.*

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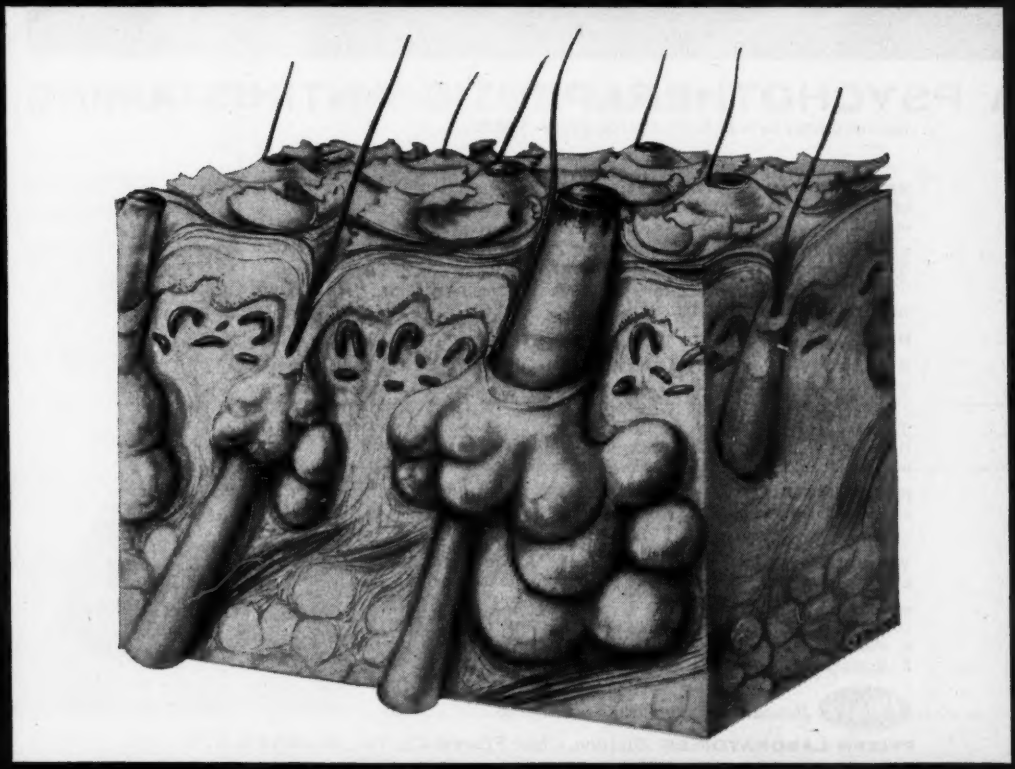
cut treatment time in half
using half the amount of vitamin A

in **acne,**

eczemas, dry scaly skin

aquasol A

the original aqueous, natural
high-potency vitamin A in capsule form



hyperkeratosis, desquamation and comedone formation with resultant follicular plugging

1 Aquasol A capsules are aqueous

— far faster, more complete absorption of their water-solubilized vitamin A (up to 300% higher blood levels as compared with oily vitamin A).

2 Aquasol A capsules contain natural vitamin A for faster, better utilization.

Natural vitamin A provides all known and fully utilizable physiologically active isomers of vitamin A — as compared with synthetic vitamin A which affords only one isomer, an isomer requiring conversion in the body before it can be utilized in certain enzyme processes.

Vitamin A has become an integral part of therapy in acne, chronic eczemas, excessively dry skin and other hyperkeratotic lesions. Why not use more effective, convenient Aquasol A capsules. Special processing of the natural vitamin A removes potential allergenic non-vitamin materials.

three separate high potencies of

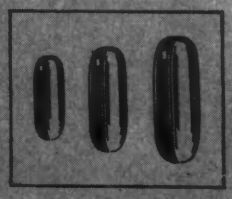
AQUASOL A CAPSULES

(water-solubilized natural vitamin A)
. . . per capsule:

25,000 U.S.P. units

50,000 U.S.P. units

100,000 U.S.P. units



bottles of 100, 500 and 1000 capsules

Samples and literature available upon request

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* Specializing in the treatment of Alcoholic and Drug Addictions. Experienced in all methods of treatment and the use of modern drugs. Treatment individualized.

* The Sanatorium is a Member of the American Hospital Association, the National Association of Private Psychiatric Hospitals and the Mississippi Hospital Association.

Specific antitussive action of Narcotine affords selective control of the cough reflex.

● non-toxic ● non-habit forming
● free from side effects

RHINALL COUGH SYRUP

makes cough more productive

soothes bronchial irritation

Supplied: 3 oz. and pint bottle. Exempt Narcotic

Each 5 cc. teaspoonful contains:

Narcotine	5.0 mg.
Phenylephrine Hydrochloride	5.0 mg.
Pyrilamine Maleate	12.5 mg.
Glyceryl Guaiacolate	50.0 mg.
Citric Acid	50.0 mg.
Sodium Citrate	22.5 mg.
Alcohol	5.0%

RHINOPTO COMPANY - Dallas, Texas

now—an antibiotic troche that

STOPS COUGH TOO

The *cough control* provided by homarylamine (a non-narcotic antitussive) approximates that of codeine.

Three antibiotics (bacitracin, tyrothricin, neomycin) act in combination against a wide variety of pathogens—with little danger of side reactions.

The anesthetic-analgesic effect of benzocaine brings *soothing relief* to inflamed tissues of mouth and throat.

PENTAZETS now extend the therapeutic usefulness of convenient troche medication. Each pleasant-tasting PENTAZETS troche acts promptly against the most bothersome aspects of mouth and throat irritations.

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Pentazets®

antitussive—antibiotic-anesthetic—analgesic troches

MSD MERCK SHARP & DOHME
DIVISION OF MERCK & CO., INC., PHILADELPHIA 1, PA.

Dosage: Three to 5 troches daily for 2 to 5 days.
Supplied: In vials of 12.
PENTAZETS is a trademark of Merck & Co., Inc.

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your

TMA LIFE PLAN

now offers

OPTIONAL BENEFITS

WITH LARGER AMOUNTS OF LIFE INSURANCE AT

OLDER AGES

TWO EXCELLENT PLANS – CHOOSE THE ONE BEST DESIGNED TO SERVE YOUR NEEDS....

INFORMATION ON BOTH TMA LIFE PLANS

Both TMA Plans (A & B) offer extremely low cost life insurance. The only difference is that Plan B offers more life insurance in the older ages for TMA members and their wives and carries a proportionately higher premium in the later years. Both plans are non-cancellable, incontestable after 2 years from date except for non-payment of premiums. Under either plan, you can convert (prior to age 60) to any program of life or endowment with no medical examination or further evidence of insurability. Both plans are renewable to attained age 71.

PLAN A

Plan A is the original TMA Life Plan with the $100.00 uniform annual premium.

Premiums are payable: $100.00 annually
$ 50.35 semi-annually
$ 25.35 quarterly
$ 8.54 monthly bank draft

PLAN B

Plan B is the plan with the new optional benefits offering larger amounts of life insurance in the later years of your life (with a proportionate premium increase). Under Plan B, your age at time of enrollment determines annual premium. (See top of next page.) You may pay your applicable premium as shown below:

Annually	$100.00	$133.33	$214.28	$244.95
Semi-annually	$ 50.35	$ 67.02	$107.49	$122.83
Quarterly	$ 25.35	$ 33.68	$ 53.67	$ 61.39
Monthly bank draft	$ 8.54	$ 11.31	$ 18.06	$ 20.61

Choice of Plan must be made at the time of original enrollment. (If you own Plan A now, you may exchange for Plan B prior to February 1, 1959.) If you desire to exchange plans, please correspond directly with the TMA Life Department, Great American Reserve Insurance Company, 2020 Live Oak Street, Dallas.

COMPARISON OF BENEFITS

PLAN A			PLAN B	
ANNUAL COST	AMT. INSURANCE	AGES	ANNUAL COST	AMT. INSURANCE
$100.00	$26,000.00	to 35*	$100.00	$26,000.00
$100.00	$15,000.00	36-45*	$100.00	$15,000.00
$100.00	$ 7,500.00	46-55*	$133.33	$10,000.00
$100.00	$ 3,500.00	56-65*	$214.28	$ 7,500.00
$100.00	$ 1,750.00	66-70*	$244.95	$ 5,000.00

*AGES INCLUSIVE • AGES TO NEAREST BIRTHDAY.

THE TMA LIFE PLAN IS A GOOD PLAN FOR YOUR WIFE TO OWN

Either Plan A or Plan B can be purchased by your wife. An individual policy is issued to the wife. The wife is eligible regardless of whether the member of TMA is enrolled. There is no difference in the policy of the TMA member or the wife. Wife's signature required on her application.

select your plan and mail your application now!

Send no money — you will be billed when your policy is issued.

APPLICATION

MAIL TO: TEXAS MEDICAL ASSOCIATION
1801 N. LAMAR BLVD. • AUSTIN

APPLICATION FOR TEXAS MEDICAL ASSOCIATION LIFE PLAN
FOR THE TMA MEMBER OR HIS WIFE
THROUGH GREAT AMERICAN RESERVE INSURANCE COMPANY

(1) Name_____
FIRST MIDDLE INITIAL LAST

(2) Permanent Mailing Address_____
STREET TOWN STATE

(3) Height_____ Weight_____ Date of Birth_____ Age_____
MONTH DAY YEAR

(4) Name of Beneficiary_____ Relationship_____
(SHOW GIVEN NAME — EXAMPLE: JANE DOE, NOT MRS. JOHN DOE)

(5) Do you know of any impairment now existing in your health or physical condition? Yes_____ No_____
Have you ever had or been treated for cancer, tuberculosis, disease of heart, high blood pressure, diabetes, epilepsy or nervous disorder? Yes_____ No_____
Have you been examined or treated by doctor during the past three years? Yes_____ No_____
Please explain fully any "yes" answer. Include date, reason, doctor's name and address.

Information in this application is given to obtain this insurance and is true and complete to the best of my knowledge and belief. The Company shall incur no obligation because of this application unless and until a policy is delivered to the applicant and the First Payment is paid in full while the health or other conditions affecting insurability of the applicant are as described in this application.

Signature of Applicant_____ Dated_____
(1020) MONTH DAY YEAR

Statement for your premium will be sent to you along with your policy. You may pay in any way you choose (please indicate mode you prefer):

ANNUALLY ☐ SEMI-ANNUALLY ☐
QUARTERLY ☐ MONTHLY ☐
(bank draft plan)

NOTE:
Application is not binding until policy has actually been received and first payment made to underwriting company.

I WANT PLAN A ☐
I WANT PLAN B ☐

In potentially-serious infections...

*TRADEMARK, REG. U. S. PAT. OFF.

†TRADEMARK, REG. U. S. PAT. OFF.—THE UPJOHN BRAND OF TETRACYCLINE

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††TRADEMARK

The Upjohn Company, Kalamazoo, Michigan

Upjohn

Make new Panalba*

(Panmycin† Phosphate plus Albamycin)**

your broad-spectrum antibiotic of first resort

effective against more than 30 common pathogens, even including resistant staphylococci.

Available forms:

1. Panalba Capsules, bottles of 16 and 100 capsules. Each capsule contains:

Panmycin phosphate (tetracycline phosphate complex) equivalent to tetracycline hydrochloride .....250 mg.
Albamycin (as novobiocin sodium)...125 mg.

2. Panalba KM†† Flavored Granules. When sufficient water is added to fill the bottle, each teaspoonful (5 cc.) contains:

Panmycin (tetracycline) equivalent to tetracycline hydrochloride .....125 mg.
Albamycin (as novobiocin calcium)..62.5 mg.
Potassium metaphosphate .....100 mg.

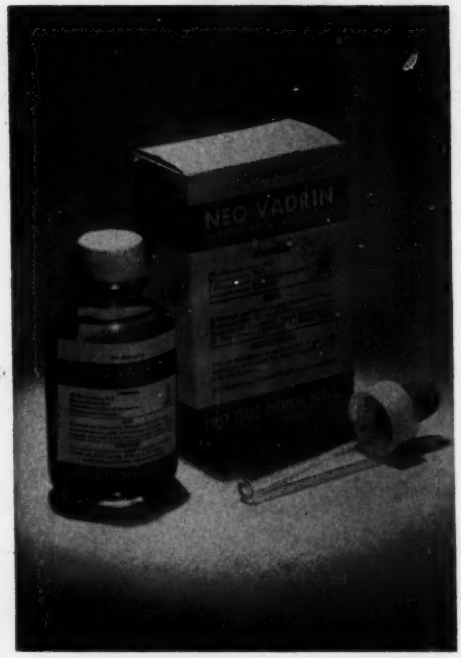
Dosage:

Panalba Capsules
Usual adult dosage is 2 capsules q.i.d.

Panalba KM Granules
For the treatment of moderately acute infections in infants and children, the recommended dosage is 1 teaspoonful per 15 to 20 lbs. of body weight per day, administered in 3 to 4 equal doses. Severe or prolonged infections require higher doses. Dosage for adults is 2 to 4 teaspoonfuls 3 or 4 times daily, depending on the type and severity of the infection.

FIRST TEXAS'

NEO-VADRIN The Ideal Nose Drop



★ Does not Burn or Cause Engorgement of the Nasal Mucosa.

★ Lasts Hours — Not Minutes.

Available:

1 oz. dropper bottle

¾ oz. spray bottle

NEO-VADRIN is a long-lasting nasal decongestant for the relief and drainage of congested nasal passages due to Head Colds and Hay Fever or other Allergies

FIRST TEXAS
CHEMICAL MANUFACTURING CO.

1903 - 1958: *Celebrating 55 years of Making* FINE PHARMACEUTICALS

1810 N. Lamar **Dallas**

FAST-ACTING ORAL BROAD-SPECTRUM THERAPY. The modern blue and yellow ACHROMYCIN V Capsules, combining equal parts of pure crystalline ACHROMYCIN Tetracycline HCl and Citric Acid, provide unsurpassed oral broad-spectrum therapy.

Speed of absorption adds new emphasis to the benefits of true broad-spectrum action, minimum side effects and wide range effectiveness that have established ACHROMYCIN as an antibiotic of choice for decisive control of infection.

REMEMBER THE V WHEN SPECIFYING ACHROMYCIN V. New blue and yellow capsules (sodium-free)—250 mg. with 250 mg. citric acid, and 100 mg., with 100 mg. citric acid.

ACHROMYCIN V dosage; Recommended basic oral dosage is 6-7 mg. per lb. body weight per day. In acute, severe infections often encountered in infants and children, the dose should be 12 mg. per lb. body weight per day. Dosage in the average adult should be 1 Gm. divided into four 250 mg. doses.

ACHROMYCIN* V CAPSULES

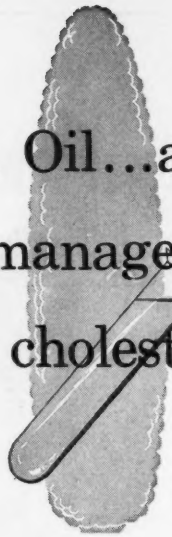
Tetracycline HCl and Citric Acid Lederle

LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Lederle

*Reg. U. S. Pat. Off.

Mazola® Corn Oil...a palatable food effective in the management and control of serum cholesterol levels



Extensive clinical tests show that when the diet contains an adequate amount of Mazola Corn Oil, serum cholesterol levels tend to be normal... high blood cholesterol levels are lowered, normal levels maintained.

Fortunately for both physician and patient, Mazola Corn Oil is not only rich in unsaturated fatty acids, it is also a delicious food. It becomes an enjoyable and normal part of the patient's daily meals—no complicated or special diet is required.

Here is a therapy easy for you to prescribe, easy and pleasant for your patients to follow.

Nutritional authorities generally recommend that fats should provide no more than 30% of the total calories. In cholesterol-lowering diets from one-third to one-half of these fats should be unsaturated, such as in Mazola Corn Oil.

IN COOKING OR SALADS

Mazola Corn Oil is a superlative cooking oil as well as a delicious salad oil. Adequate amounts can be eaten daily—in a wide variety of salad dressings and in a great number of fried and baked foods.

MOST EFFECTIVE

Pure, clear, bland and odorless. Mazola Corn Oil is stable and dependable, providing the full measure of cholesterol-lowering unsaturated fatty acids characteristic of corn oil.

ECONOMICAL

Mazola Corn Oil is sold in grocery stores throughout the country, is available everywhere. Its comparatively low cost makes it as economical as it is effective.

MAZOLA* CORN OIL is a rich source of unsaturated fatty acids. It can form a regular part of the diet without major changes in eating habits to provide an effective unsaturated oil as a part of the daily meals.

EACH TABLESPOONFUL OF MAZOLA CORN OIL PROVIDES NOT LESS THAN:

Linoleic Acid	7.4 Gm.
Sitosterols	130 mg.
Natural Tocopherols	15 mg.

TYPICAL AMOUNTS PER DIET

For a 3600 calorie diet	3 tablespoonsful
For a 3000 calorie diet	2.5 tablespoonsful
For a 2000 calorie diet	1.5 tablespoonsful

*Reg. U. S. Pat. Off.

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KASTOFF®

the modern method for removing casts!

KASTOFF . . . the new way to remove casts quickly, painlessly, safely. NOW . . . with the KASTOFF method even your technician can easily remove casts of the hardest materials without producing the fear, cuts, or burns among your patients of old fashioned cast removal methods.

KASTOFF comes complete with turning clips and one dozen wires at $27.00, with additional wires and clips for $9.50 per dozen. Write for illustrated booklet outlining the use of KASTOFF. Names of successful users of KASTOFF will be furnished upon request.

Call your surgical supply man or write:

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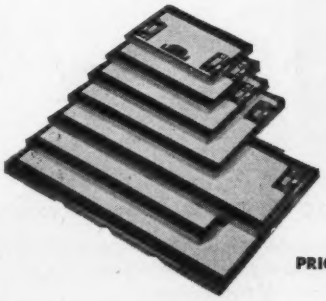
★ QUICKLY
★ ECONOMICALLY
★ EASILY
★ PAINLESSLY
★ SAFELY

Use Kastoff on your next cast. You and your patient will be glad you did.

PRICE COMPLETE **$27.00**
WIRES PER DOZEN **9.50**

G-E molded cassettes cost less— last far longer!



Molded-rubber frame cushions jolts, keeps front and back of cassette in *true alignment*. Built-in glass-fiber pad gently squeezes screens and film for uniform contact always. "Slide-easy" latches release at light finger pressure, resist accidental opening. Molded-rubber seal prevents entry of light. Exclusive rubber hinge — thoroughly proved in ½-million flexings that left it *bonded as firmly as at time of manufacture!*

PRICES: 5x7—$14.00 6½x 8½—$16.50 8x10—$18.00 11x14—$23.25 7x17—$23.50 10x12—$20.00 14x17—$25.25

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Next door to Great Southwest development between Dallas and Fort Worth, Texas.

Anticipated increase of 100,000 population in trade territory within the next seven years.

Will arrange offices or suites to suit your needs—8,000 square feet available on or about April 1, 1959.

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A complete nutritional supplement designed for the needs of the pregnant woman.

ERUCYTE OB provides:

Absorbable calcium (phosphorus-free) to combat leg cramps and other indications of hypocalcemia.
Ferrous fumarate, the highly effective, well-tolerated hematinic to provide hemoglobin for mother and developing child.
Adequate vitamins to combat the stresses and requirements of pregnancy.
Needed trace minerals to ensure optimal cellular function.

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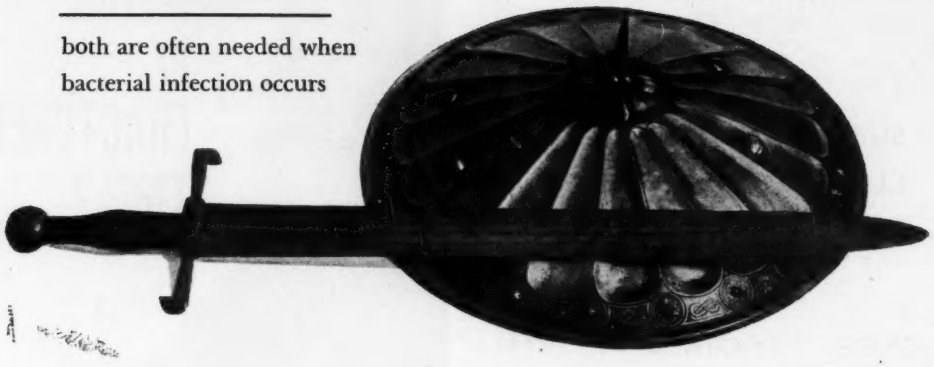
LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

■ prompt, aggressive
antibiotic action
■ a reliable defense against
monilial complications

both are often needed when
bacterial infection occurs



for a direct strike at infection
Mysteclin-V contains tetracycline phosphate complex

It provides a direct strike at all tetracycline-susceptible organisms (most pathogenic bacteria, certain rickettsias, certain large viruses, and Endamoeba histolytica).

It provides the new chemical form of the world's most widely prescribed broad spectrum antibiotic.

It provides unsurpassed initial blood levels – higher and faster than older forms of tetracycline – for the most rapid transport of the antibiotic to the site of infection.

for protection against monilial complications
Mysteclin-V contains Mycostatin

It provides the antifungal antibiotic, first tested and clinically confirmed by Squibb, with specific action against Candida (Monilia) albicans.

It acts to prevent the monilial overgrowth which frequently occurs whenever tetracycline or any other broad spectrum antibiotic is used.

It protects your patient against antibiotic-induced intestinal moniliasis and its complications, including vaginal and anogenital moniliasis, even potentially fatal systemic moniliasis.

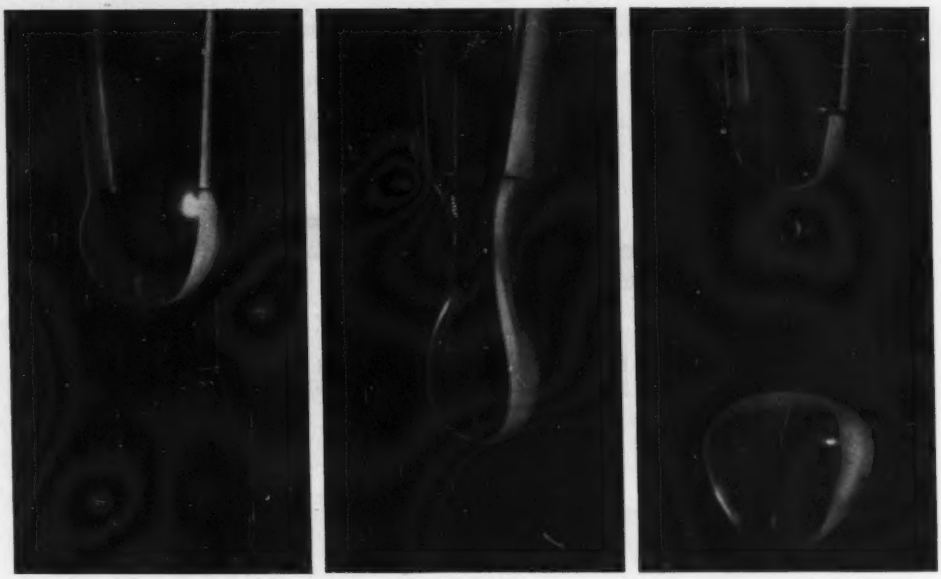
MYSTECLIN-V

Squibb Tetracycline Phosphate Complex (Sumycin) and Nystatin (Mycostatin)

Capsules *(250 mg./250,000 u.), bottles of 16 and 100.* Half-strength Capsules *(125 mg./125,000 u.), bottles of 16 and 100.*
Suspension *(125 mg./125,000 u. per 5 cc.) 60 cc. bottles.* Pediatric Drops *(100 mg./100,000 u. per cc.). 10 cc. dropper bottles.*

SQUIBB *Squibb Quality — the Priceless Ingredient*

'MYSTECLIN'®, 'SUMYCIN'®, AND 'MYCOSTATIN'® ARE SQUIBB TRADEMARKS



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ZANCHOL®

Improves Flow and Color of Bile

Zanchol (brand of florantyrone), a distinct chemical entity unrelated to the bile salts, provides the medical profession with a new and potent hydrocholeretic for treating disorders of the biliary tract.

The high degree of therapeutic activity of this new compound and its negligible side reactions yield distinct clinical advantages.

- Zanchol produces a bile low in sediment.
- Zanchol enhances the abstergent quality of bile.
- Zanchol produces a deep, brilliant green bile, regardless of its original color, suggesting improved hepatic function.
- Zanchol improves the flow and quantity of bile without increasing total bile solids.

Bile with these qualities minimizes biliary stasis, reduces sediment and debris in the bile ducts and discourages the ascent of infection.

For these reasons ZANCHOL has shown itself to be a highly valuable agent in chronic cholecystitis, cholangitis and care of patients following cholecystectomy.

Administration: One tablet three or four times a day. Zanchol is supplied in tablets of 250 mg. each. G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

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COMPREHENSIVE CONTROL OF CONSTIPATION

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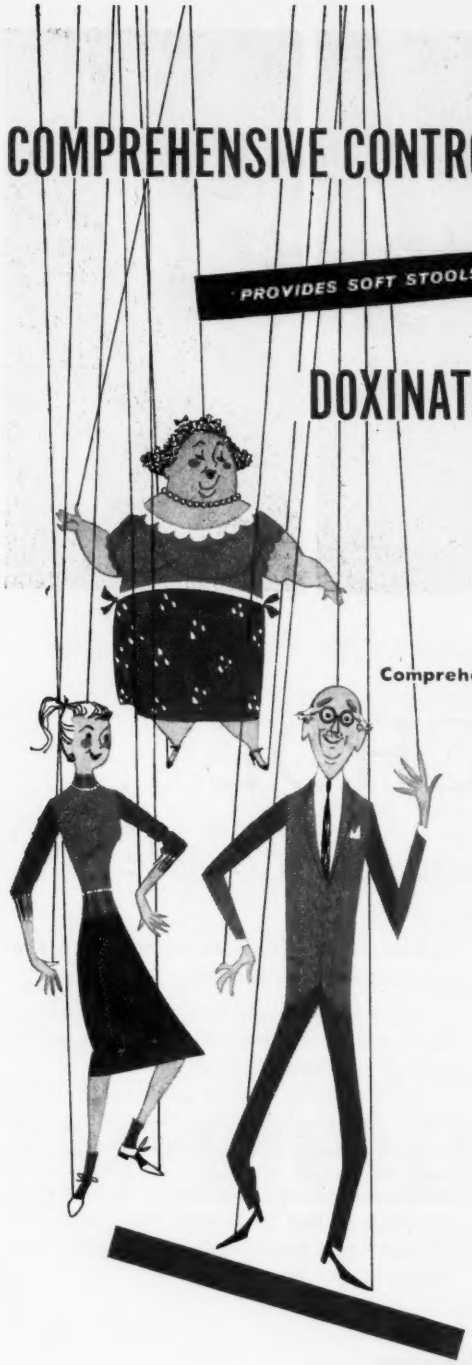

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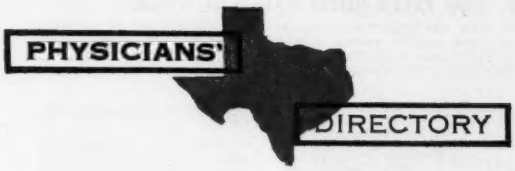

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WANTED—The following specialists to join our group in Dallas on independent basis; E N T man, Ophthalmologist, Dermatologist, Proctologist, Orthodontist. The group at present time consists of two internists, three pediatricians, urologist, surgeon, orthopedist, and two OB and GYN men and one gynecologist. All men must have their boards or be board eligible. Address Box 909, care of Texas Medical Association, 1801 North Lamar Blvd., Austin, Texas.

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WANTED—Pediatrician to join or lease space in new Clinic in suburban north east Dallas. Write Box 215, Texas State Journal of Medicine, 1801 North Lamar Blvd., Austin, Texas.

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WELL QUALIFIED general practitioner, middle age, married, family, desires relocation preferably as associate to internist but would consider group, industrial or other location with good opportunity. Contact Box 219, Texas State Journal of Medicine, 1801 North Lamar Blvd., Austin, Texas.

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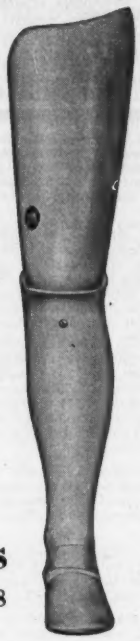

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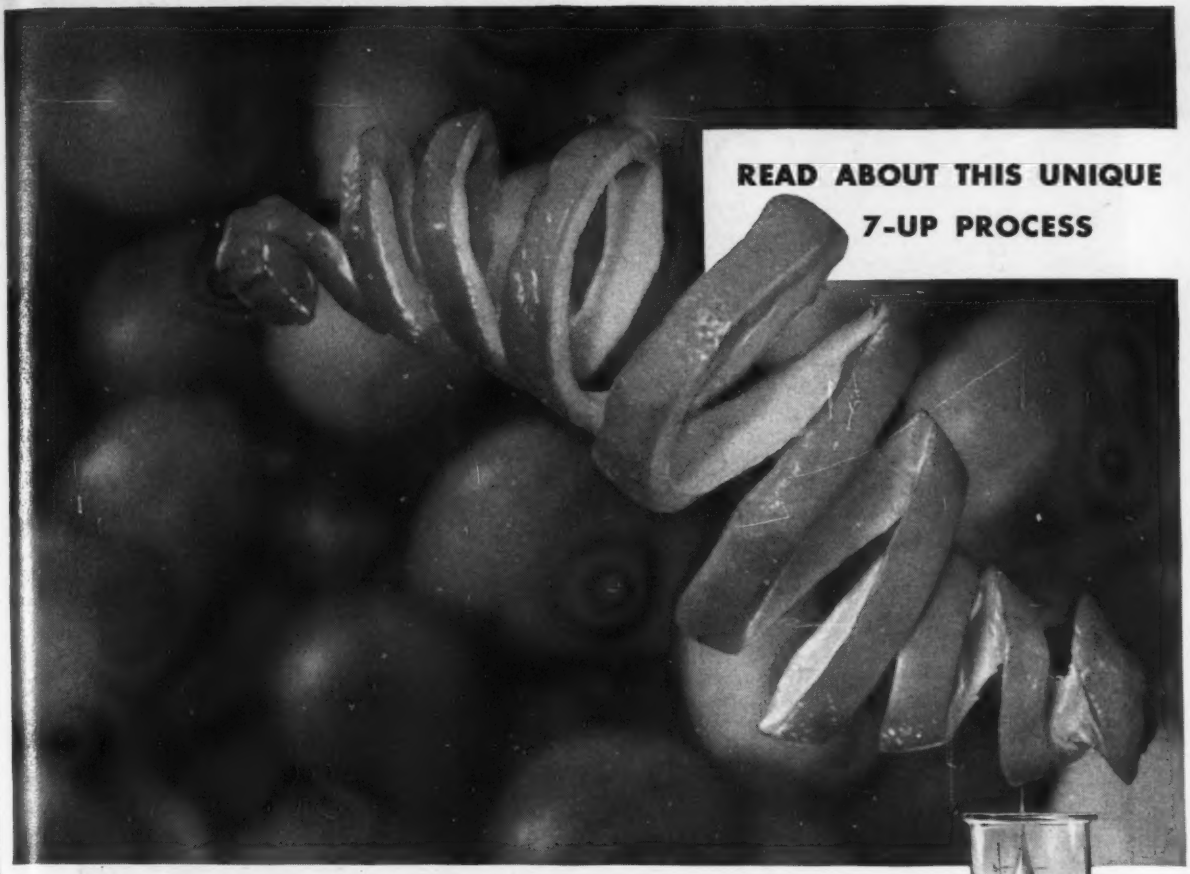

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Demerol hydrochloride	30 mg. (½ grain)

Average Dose:

1 or 2 tablets.

Narcotic blank required.

Potentiated Pain Relief

WINTHROP LABORATORIES
New York 18, N. Y. • Windsor, Ont.

Demerol (brand of meperidine), trademark reg. U.S. Pat. Off.

to relieve edema of

DIURIL®"

CHLOROTHIAZIDE

FINNERTY, F. A., Buchholz, J. H. and Tuckman, J.: J.A.M.A. **166**:141, Jan. 11, 1958.

DIURIL (Chlorothiazide) given alone to 85 patients, ". . . caused an excellent diuresis, with reduction of edema, weight, blood pressure, and albuminuria. . . . The average effective dose was found to be 1 Gm. per day by mouth. . . . The usually excellent response coupled with the absence of significant toxicity and lack of development of drug resistance makes chlorothiazide ideal for the prevention and treatment of toxemia."

DOSAGE: one or two 500 mg. tablets of DIURIL once or twice a day.

SUPPLIED: 250 mg. and 500 mg. scored tablets DIURIL (chlorothiazide); bottles of 100 and 1,000.

DIURIL is a trademark of Merck & Co., Inc.

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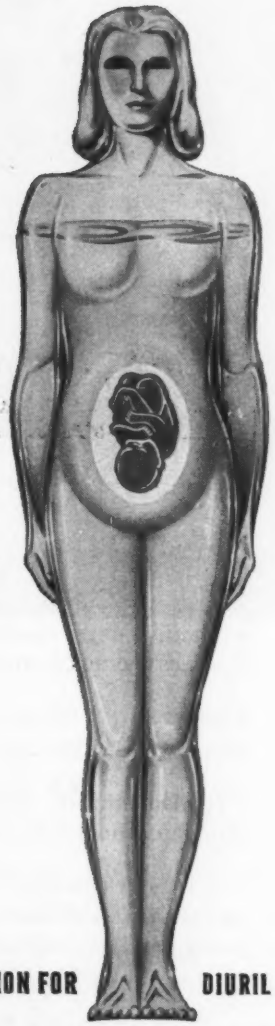
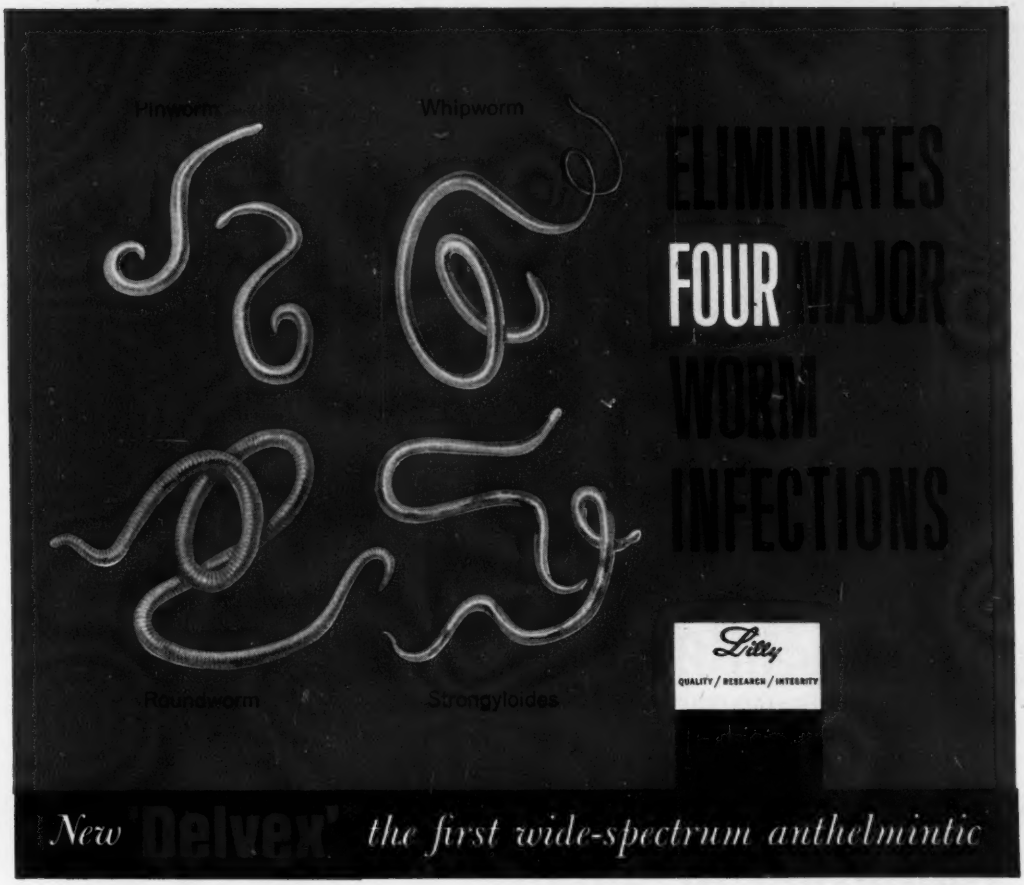
MERCK SHARP & DOHME Division of MERCK & CO., INC., Philadelphia 1, Pa. MSD

pregnancy

...caused an excellent diuresis, with reduction of edema, weight, blood pressure, and albuminuria...."



ANY INDICATION FOR DIURESIS IS AN INDICATION FOR DIURIL



Clinical studies[1] *show:*

- 'Delvex' is effective orally, usually within five days, against four of the five most common worm infections:

Pinworm	Whipworm
Roundworm	Strongyloidiasis

- It also inhibits, and sometimes eliminates, hookworm infection.
- It is fully effective in both single and multiple infections and in both heavy and light infections.
- It eliminates pinworm infection in 100 percent of patients.
- It is the first effective and practicable agent for the oral treatment of strongyloidiasis and whipworm infection.
- No adjunctive measures are needed with 'Delvex' therapy.

Further information and clinical reports may be obtained from your Lilly representative or by writing to our Medical Department.

*'Delvex' (Dithiazanine Iodide, Lilly)
1. Swartzwelder, J. C., et al.: J. A. M. A., 165:2063, 1957.

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If you were to examine these patients

could you detect the uveitis patient on Medrol*?

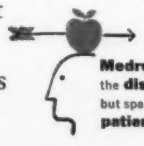
Probably not. Not without a history.

First, because he's more than likely symptom-free.

Second, because he shows none of the disturbing changes in appearance, behavior or metabolism sometimes associated with corticotherapy.

Even your practiced clinical eye would find it difficult to spot someone else's Medrol patient.

But in your own patients, you could see the advantages of Medrol right away. Why not try it?



Medrol hits the **disease,** but spares the **patient**

Upjohn

The Upjohn Company, Kalamazoo, Michigan

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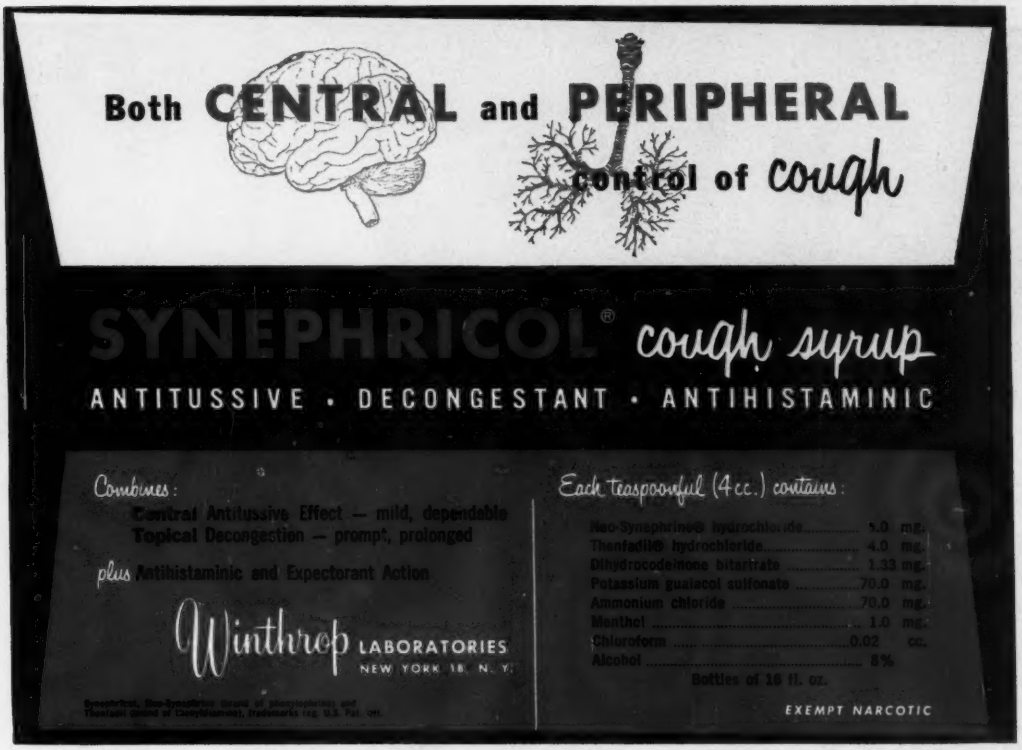

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†warts; moles; sebaceous cysts; benign tumors; wounds; lacerations; biopsies; tying superficial varicose veins; minor rectal surgery; simple fractures; compound digital injuries (not involving tendons, nerves or bones)

*U.S. PAT. NO. 2,441,498 MADE IN U.S.A.

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Director

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IN YOUR office, hospital or clinic, *more* hands powered by *more* brains are the answer of choice to the bottleneck of overwork for men of known competence.

Such men today are *building up* their stature and *cutting down* time consuming routine by selecting suitable associates and chiefs of departments, then delegating duties. The procedure—simply: (1) dictate a letter to us describing the position and qualifications; (2) pick up the telephone and outline the situation person-to-person *or* (3) walk into our offices and say: "Please show me the records of a few candidates qualified to carry a share of my load without wincing."

Result: You will reach *more* qualified candidates, *speed up* your selection of a satisfactory co-worker . . . May we explore the field for you *now*?

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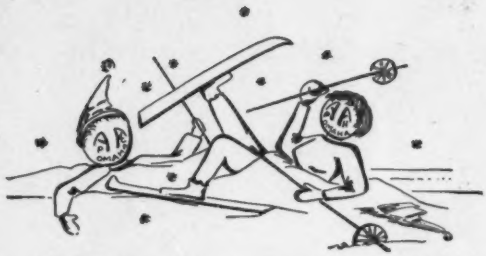

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Not every physician needs the exquisite accuracy obtainable with the new Birtcher Electrocardiograph.

At present probably only about one physician in several hundred is sufficiently versed in cardiographic interpretation to appreciate the extraordinary refinements of cardiac electrical activity recording, and thus to comprehend the real character of this instrument's traces.

Fewer still are the number of those physicians, qualified by training in physics and engineering, to understand the functioning of so complex an electronic device as the direct writing Electrocardiograph.

The Birtcher was conceived and executed to surpass the most rigid specifications set up by various august cardiological bodies and governmental groups.

Not a single compromise has been made in accuracy of this direct writing instrument, in order to gain quick acceptance and promote rapid sales. Not one iota of known engineering for accuracy has been prostituted to satisfy those "customers" whose apparent requirements are based on a lack of knowledge or actual false information. Not a single sacrifice in engineering integrity has been made to cater to the salesman . . . nor for the sake of a lower price.

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The Birtcher Electrocardiograph, presented with courage and dignity, engendered by integrity to provide a professional lifetime of dependable, accurate cardiography, anticipating new methods and concepts in cardiology and cardiography yet to come, costs somewhat more . . . Yet, in four short months since its introduction, it has proven itself the hard way from coast to coast and in about twenty foreign countries as well, and it is firmly established among quite a number of those most eminent in cardiography.

Although the Birtcher is purveyed through surgical supply dealers, only those who, through special experience and training in such electronic devices and are otherwise qualified to render service, have been selected to have the Birtcher franchise.

A physician, by simple request on his letterhead or Rx blank, may receive without further ado, a large, five color brochure descriptive of the Birtcher Electrocardiograph and, as a gift, a new, special dual speed ECG measuring rule, together with a new booklet on cardiographic diagnosis, both especially prepared for Birtcher by a distinguished cardiographer. Please address your request to: The Birtcher Corporation, Department TS-1258, 4371 Valley Boulevard, Los Angeles 32, California.

THE RATIONALE FOR THE USE OF VITAMINS IN FORESTALLING INFECTIONS

Many clinicians believe that good nutrition plays a significant role in preventing bacterial infections, and that immunity depends on adequate vitamin levels. Tisdall[1] states that "a low intake of a number of vitamins, a low intake of minerals, and a change in the quality of protein can all lower resistance to infection."

Other studies show the important role of the B vitamins in antibody formation. Thus, *Nutrition Reviews*[2] reports: "Present evidence indicates that certain B vitamins, notably pyridoxine, pantothenic acid and folacin, play a significant role in antibody synthesis." According to Pollack and Halpern,[3] "Under-nutrition leads to increased susceptibility to infection and decreased resistance to established disease." And "vitamin deficiency states also may adversely influence circulating antibodies."

Halpern[4] reports that "good nutrition is important for optimal resistance to infection, for a superior tissue capability to cope with disease and injury, and for maximum antibody production . . . nutrition participates in the prophylaxis against most acute infections . . ."

And while MacBryde[5] feels that evidence is lacking to support the view that a higher than normal intake of vitamins will improve resistance to infection, he also states: "Restoration of nutrition to normal exerts a favorable influence on practically all disease conditions . . . Often the outcome will depend more upon the correction of the malnutrition than upon any therapy directed toward the malady."

THERAGRAN

SQUIBB VITAMINS FOR THERAPY

now expanded to include additional essential vitamins—

and at no extra cost to your patients

Each Theragran Capsule supplies:

Vitamin A	25,000 U.S.P. units
Vitamin D	1,000 U.S.P. units
Thiamine Mononitrate	10 mg.
Riboflavin	10 mg.
Niacinamide	100 mg.
Ascorbic Acid	200 mg.
Pyridoxine Hydrochloride	5 mg.
Calcium Pantothenate	20 mg.
Vitamin B_{12} Activity Concentrate	5 mcg.

Dosage: 1 or more capsules daily as indicated.

Supply: Family Packs of 180. Bottles of 30, 60, 100 and 1,000.

Also Available: THERAGRAN Liquid, bottles of 4 ounces; THERAGRAN Junior bottles of 30 and 100 capsules; and THERAGRAN-M (Squibb Vitamin-Minerals for Therapy), bottles of 30, 60, 100 and 1,000 capsule-shaped tablets.

References: 1. Tisdall, F. F.: Clinical Nutrition, ed. by Joliffe, N.; Tisdall, F. F., and Cannon, P. R.: Paul B. Hoeber, Inc., New York, 1950, p. 748. 2. Nutrition Reviews, *15*:47, (Feb.) 1957. 3. Pollack, H., and Halpern, S. L.: Therapeutic Nutrition, National Academy of Sciences and National Research Council, Washington, D. C., 1952, p. 18. 4. Halpern, S. L.: Ann. N. Y. Acad. Science *63*:147, (Oct. 28) 1955. 5. MacBryde, C. N.: Signs and Symptoms, J. B. Lippincott Co., Phila., 3rd Ed. 1957, p. 818.

SQUIBB *Squibb Quality—The Priceless Ingredient*

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Many such hypertensives have been on *Rauwiloid* for 3 years and more*

for Rauwiloid IS better tolerated... "alseroxylon [Rauwiloid] is an antihypertensive agent of equal therapeutic efficacy to reserpine in the treatment of hypertension but with significantly less toxicity."

*Ford, R.V., and Moyer, J.H.: Rauwolfia Toxicity in the Treatment of Hypertension, Postgrad. Med. 23:41 (Jan.) 1958.

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just two tablets at bedtime

After full effect one tablet suffices

Enhances safety when more potent drugs are needed.

Rauwiloid® + Veriloid®
alseroxylon 1 mg. and alkavervir 3 mg.
for moderate to severe hypertension.
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alseroxylon 1 mg. and hexamethonium chloride dihydrate 250 mg.
in severe, otherwise intractable hypertension. Initial dose, ½ tablet q.i.d.

Both combinations in convenient single-tablet form.

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Relieve moderate or severe pain

Reduce fever

Alleviate the general malaise of upper respiratory infections

'TABLOID'

'EMPIRIN' COMPOUND®

WITH

CODEINE PHOSPHATE*

maximum codeine analgesia/optimum antipyretic action

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Symbols OF PROVEN PAIN RELIEF

gr. 1

gr. ½

gr. ¼

gr. ⅛

Formulas for dependable relief...

...from moderate to severe pain complicated by tension, anxiety and restlessness.

'CODEMPIRAL'® NO. 3*

Codeine Phosphate . . . . . gr. ½
Phenobarbital . . . . . gr. ¼
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Codeine Phosphate . . . . . gr. ¼
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...from pain of muscle and joint origin, simple headache, neuralgia, and the symptoms of the common cold.

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...from mild pain complicated by tension and restlessness.

'EMPIRAL'®

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TETRACYCLINE-ANTIHISTAMINE-ANALGESIC COMPOUND LEDERLE

A versatile, well-balanced formula for treating common upper respiratory infections, particularly during respiratory epidemics; when bacterial complications are observed or are likely; when patient's history is positive for recurrent otitic, pulmonary, nephritic, or rheumatic involvement.

CHECKS SYMPTOMS: Includes traditional components for rapid relief of the traditional nonspecific nasopharyngitis, symptoms of malaise, chilly sensations, inconstant low-grade fever, headache, muscular pain, pharyngeal and nasal discharge.

Available on prescription only.

Adult dosage for ACHROCIDIN Tablets and new caffeine-free ACHROCIDIN Syrup is two tablets or teaspoonfuls of syrup three or four times daily. Dosage for children according to weight and age.

TABLETS *(sugar coated)*

Each Tablet contains:

ACHROMYCIN® Tetracycline	125 mg.
Phenacetin	120 mg.
Caffeine	30 mg.
Salicylamide	150 mg.
Chlorothen Citrate	25 mg.

Bottles of 24 and 100.

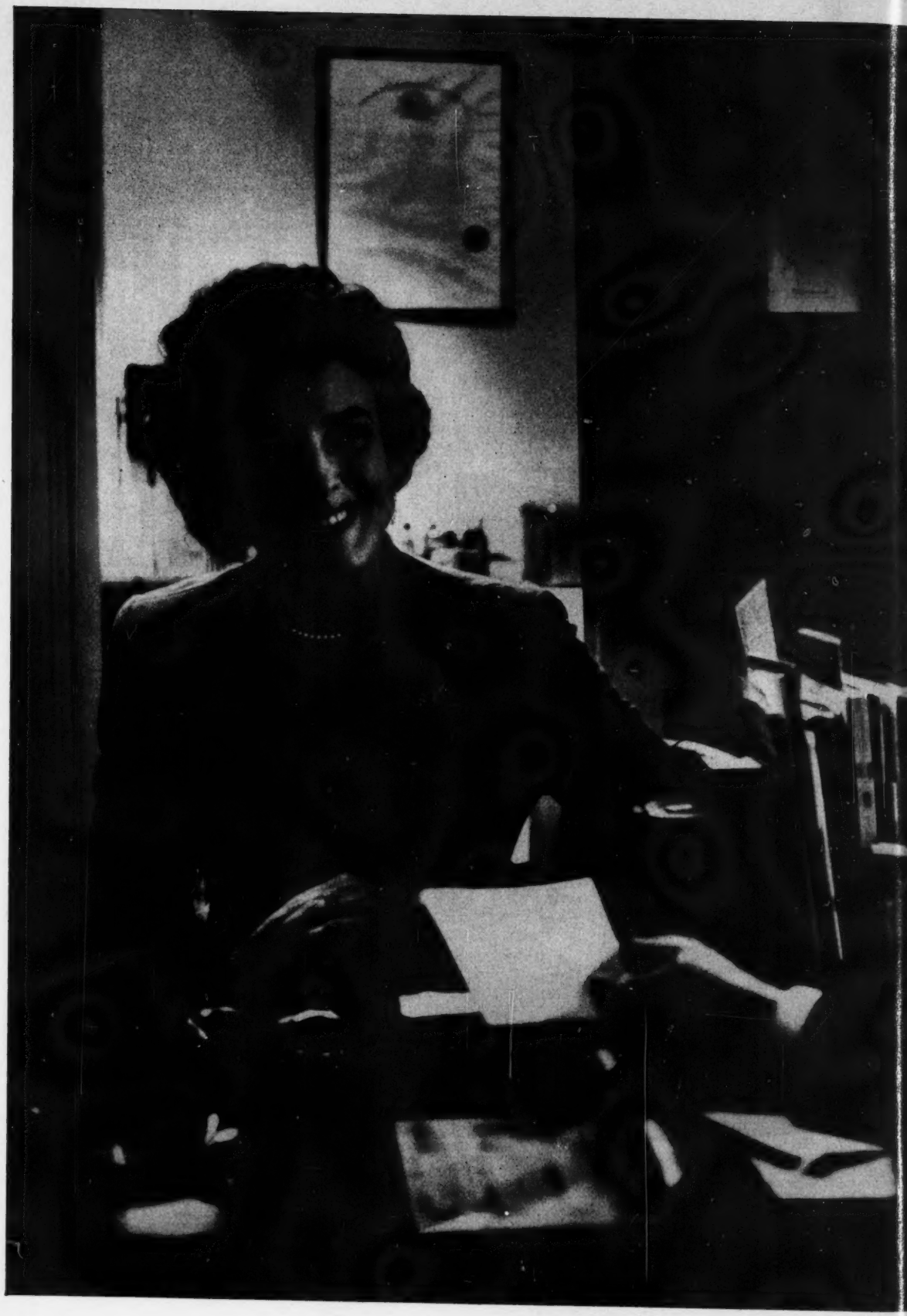
SYRUP *(lemon-lime flavored)*

Each teaspoonful (5 cc.) contains:

ACHROMYCIN® Tetracycline equivalent to tetracycline HCl	125 mg.
Phenacetin	120 mg.
Salicylamide	150 mg.
Ascorbic Acid (C)	25 mg.
Pyrilamine Maleate	15 mg.
Methylparaben	4 mg.
Propylparaben	1 mg.

Bottle of 4 oz.

LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York



"Much better– thank you, doctor"

Proven in research

1. Highest tetracycline serum levels
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And now in practice

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COSA-TETRACYN*

GLUCOSAMINE-POTENTIATED TETRACYCLINE

CAPSULES
(black and white)
250 mg., 125 mg.
(for pediatric or long-term therapy)

ORAL SUSPENSION
(orange-flavored)
125 mg. per tsp. (5 cc.)
2 oz. bottle

NEW! PEDIATRIC DROPS
(orange-flavored) 5 mg. per drop, calibrated dropper, 10 cc. bottle

COSA-TETRASTATIN*

glucosamine-potentiated tetracycline with nystatin

Antibacterial plus added protection against monilial super-infection

CAPSULES (black and pink) 250 mg. Cosa-Tetracyn (with 250,000 u. nystatin)

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COSA-TETRACYDIN*

glucosamine-potentiated tetracycline-analgesic-antihistamine compound

For relief of symptoms and malaise of the common cold and prevention of secondary complications

CAPSULES (black and orange)—each capsule contains: Cosa-Tetracyn 125 mg.; phenacetin 120 mg.; caffeine 30 mg.; salicylamide 150 mg.; buclizine HCl 15 mg.

REFERENCES: 1. Carlozzi, M.: Antibiotic Med. & Clin. Therapy 5:146 (Feb.) 1958. 2. Welch, H.; Wright, W. W., and Staffa, A. W.: Antibiotic Med. & Clin. Therapy 5:52 (Jan.) 1958. 3. Marlow, A. A., and Bartlett, G. R.: Glucosamine and leukemia, Proc. Soc. Exp. Biol. & Med. 84:41, 1953. 4. Shalowitz, M.: Clin. Rev. 1:25 (April) 1958. 5. Nathan, L. A.: Arch. Pediat. 75:251 (June) 1958. 6. Cornbleet, T.; Chesrow, E., and Barsky, S.: Antibiotic Med. & Clin. Therapy 5:328 (May) 1958. 7. Stone, M. L.; Sedlis, A., Bamford, J., and Bradley, W.: Antibiotic Med. & Clin. Therapy 5:322 (May) 1958. 8. Harris, H.: Clin. Rev. 1:15 (July) 1958.

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for depression

▲Deprol▲†

Clinically confirmed in over 2,500 documented case histories[1,2]

CONFIRMED EFFICACY

Deprol ► acts promptly to control depression *without* stimulation

► restores natural sleep

► reduces depressive rumination and crying

DOCUMENTED SAFETY

Deprol is unlike amine-oxidase inhibitors

► does not adversely affect blood pressure or sexual function

► causes no excessive elation

► produces no liver toxicity

► does not interfere with other drug therapies

Deprol is unlike central nervous stimulants

► does not cause insomnia

► produces no amphetamine-like jitteriness

► does not depress appetite

► has no depression-producing aftereffects

► can be used freely in hypertension and in unstable personalities

Dosage: Usual starting dose is 1 tablet q.i.d. When necessary, this dose may be gradually increased up to 3 tablets q.i.d.

Composition: Each tablet contains 400 mg. meprobamate and 1 mg. 2-diethylaminoethyl benzilate hydrochloride (benactyzine HCl).

Supplied: Bottles of 50 scored tablets.

1. Alexander, L.: Chemotherapy of depression—Use of meprobamate combined with benactyzine (2-diethylaminoethyl benzilate) hydrochloride. J.A.M.A. 166:1019, March 1, 1958. 2. Current personal communications; in the files of Wallace Laboratories.

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Literature and samples on request WALLACE LABORATORIES, *New Brunswick, N. J.*

THE NEW YORK POLYCLINIC

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A two-months combined surgical course comprising general surgery, traumatic surgery, abdominal surgery, gastroenterology, proctology, gynecological surgery, urological surgery. Attendance at lectures, witnessing operations, examination of patients preoperatively and post-operatively, and follow-up in the wards post-operatively. Pathology, radiology, physical medicine, anesthesia. Cadaver demonstrations in surgical anatomy, thoracic surgery, proctology, orthopedics. Operative surgery and operative gynecology on the cadaver; attendance at departmental and general conferences.

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March 2, 3, 4, and 5, 1959

Palmer House, Chicago

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The Chicago Medical Society Annual Clinical Conference should be a MUST on the calendar of every physician. Plan now to attend and make your reservation at the Palmer House.

Exactly how does new Halodrin* restore the "premenopausal prime" in postmenopausal women?

Webster defines "prime" as the period of greatest health, strength, and beauty. In a woman, these are the childbearing years between puberty and menopause—the years when her hormone production is highest.

The inevitable reduction in this hormone production as she enters the menopause often results in physical discomfort in the form of hot flushes, nervousness, insomnia, or a multiplicity of other symptoms with which you are familiar. Superimposed on this physical picture is the psychic trauma brought on by this unavoidable evidence of aging. The thing that brings her to a physician is simply that she "feels bad."

You can't make her 35 again—but the odds are good that you can make her feel like it! The secret is a combination of reassurance and hormones. The exact form and amount of the former defy objective analysis, but the latter can now be provided with scientific precision. Reduced to essentials, here is the explanation of exactly how hormones—in the form of Upjohn's new Halodrin—restore the "premenopausal prime."

The normal premenopausal woman excretes estrogens in the urine in the form of estradiol, estrone, and estriol, in an approximate 28-day average ratio of 39:15:46. Starting with this urinary excretion of estrogens, it is possible to calculate backwards and estimate the amount of estradiol that must have been secreted endogenously in order to produce these urinary levels. This is possible because the proportion of estrogens which appears in the urine following parenteral administration has been established in castrated women.

On this basis, the average endogenous output of estrogens is about 160 micrograms per day during a menstrual cycle, and 80 micrograms per day in postmenopausal women (see chart opposite). Therefore, the restoration of the "premenopausal prime" in the postmenopausal woman requires the replacement of approximately the equivalent of the 80 micrograms of estradiol per day that she no longer secretes endogenously.

Oral ethinyl estradiol is about 2 to 2½ times as potent as parenteral estradiol. Therefore, the replacement of 80 micrograms of endogenous estradiol production per day is accomplished by the oral administration of 32 to 40 micrograms of ethinyl estradiol per day.

Each Halodrin tablet contains 20 micrograms of ethinyl estradiol, which means that the recommended dosage of 2 tablets per day provides 40 micrograms of ethinyl estradiol. This offsets the loss of 80 micrograms of endogenous estradiol production in the menopausal woman; i.e., restores the "premenopausal prime."

Each Halodrin tablet also contains 1 mg. of Upjohn-developed Halotestin* (fluoxymesterone)—the most potent oral androgen known. The primary purpose is to "buffer" the ethinyl estradiol just enough to prevent breakthrough bleeding, which is obviously undesirable in the menopause. It also exerts other beneficial hormonal effects, one of which, in common with ethinyl estradiol, is a powerful anabolic action so desirable in patients of advanced years.

Upjohn

*TRADEMARK, REG. U. S. PAT. OFF.

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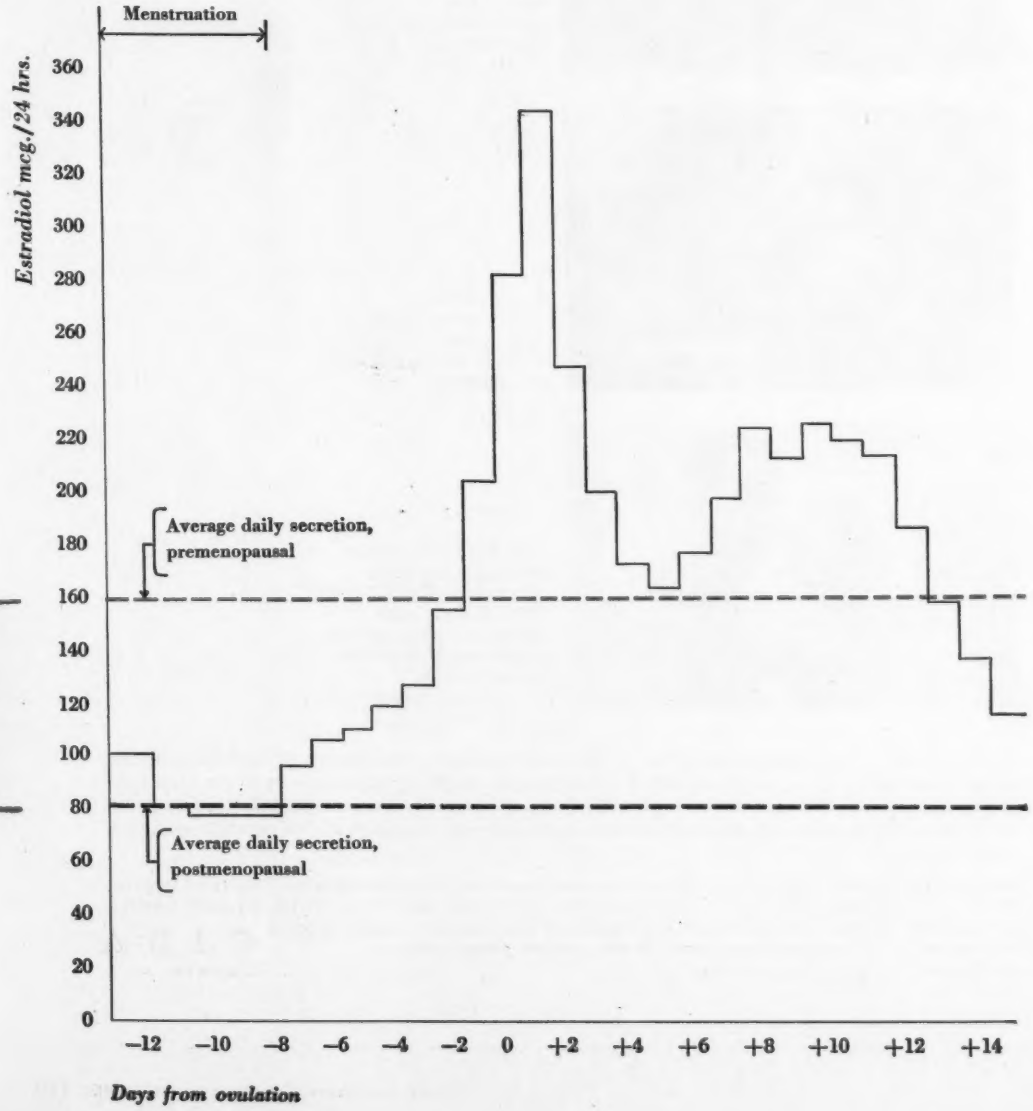
Endogenous estrogen secretion (mcg./24 hours)
(calculated from average 24-hour urinary excretion of estradiol, estrone, and estriol)

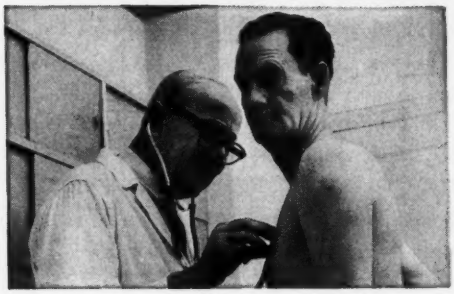


Pyribenzamine® EXPECTORANT breaks up cough

even persistent cough

Patient, factory worker, age 43, had suffered for months with persistent, dry cough, which he termed "smoker's hack."

Cough frequently interrupted his sleep, causing him to be nervous, irritable; his job efficiency was impaired.



Chest X-ray was negative and the plant physician prescribed PYRIBENZAMINE EXPECTORANT with Ephedrine. Patient noticed almost immediate relief—a week later felt "considerably better."

Pyribenzamine Expectorant with Ephedrine provides a unique combination of antitussive agents, which work three ways at once to break up the persistent cough: *Pyribenzamine* relieves histamine-induced congestion throughout the respiratory tract; *ephedrine* relaxes the bronchioles and makes breathing easier; *ammonium chloride* liquefies mucus, relieving dry cough and promoting productive expectoration.

Supplied: Pyribenzamine *Expectorant with Ephedrine*, containing 30 mg. Pyribenzamine citrate (equivalent to 20 mg. Pyribenzamine hydrochloride), 10 mg. ephedrine sulfate and 80 mg. ammonium chloride per 4-ml. teaspoon.

Also available: Pyribenzamine *Expectorant with Codeine and Ephedrine*, same formula as above with the addition of 8 mg. codeine phosphate per 4-ml. teaspoon (exempt narcotic).

CIBA
SUMMIT, N. J.

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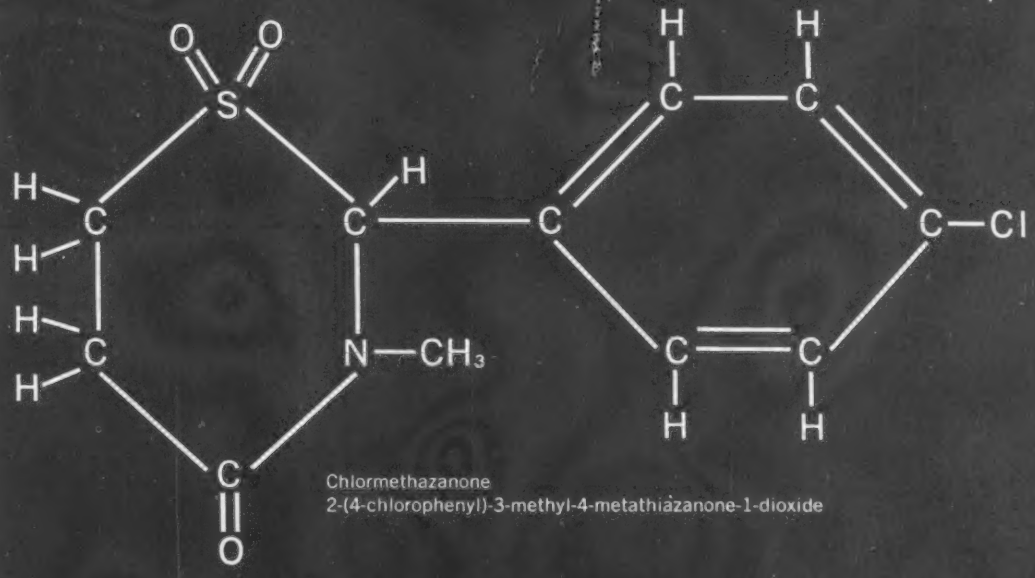
a completely new major chemical contribution to therapeutics



Chlormethazanone
2-(4-chlorophenyl)-3-methyl-4-metathiazanone-1-dioxide

unrelated chemically to any other drug in current use

designed to be equally effective as <u>*both*</u>

a MUSCLE RELAXANT

a TRANQUILIZER

Trancopal

*the first true "TRANQUILAXANT"**

offering new freedom for your patients... from muscle spasm, from tension and anxiety, from side effects

* **tran-qui-lax-ant** (tran'kwi-lak'sant) [< L. tranquillus, quiet; L. laxare, to loosen, as the muscles]

EXCEEDS OLDER DRUGS UP TO 4 TIMES IN PERCENTAGE OF CLINICAL EFFICACY (Lichtman)

The results of clinical studies of over 4000 patients by 105 physicians demonstrate that TRANCOPAL often is effective when other drugs have failed. From these studies it is clear that TRANCOPAL probably can provide more help for a greater number of tense, spastic, and/or emotionally upset patients than any other chemotherapeutic agent in current use.

TRANCOPAL... the first true "tranquilaxant"

Both a muscle relaxant and a calmative agent.

In musculoskeletal disorders, 91 per cent effective.

In anxiety and tension states, 93 per cent effective.

Lower incidence of side effects than with zoxazolamine, methocarbamol or meprobamate.

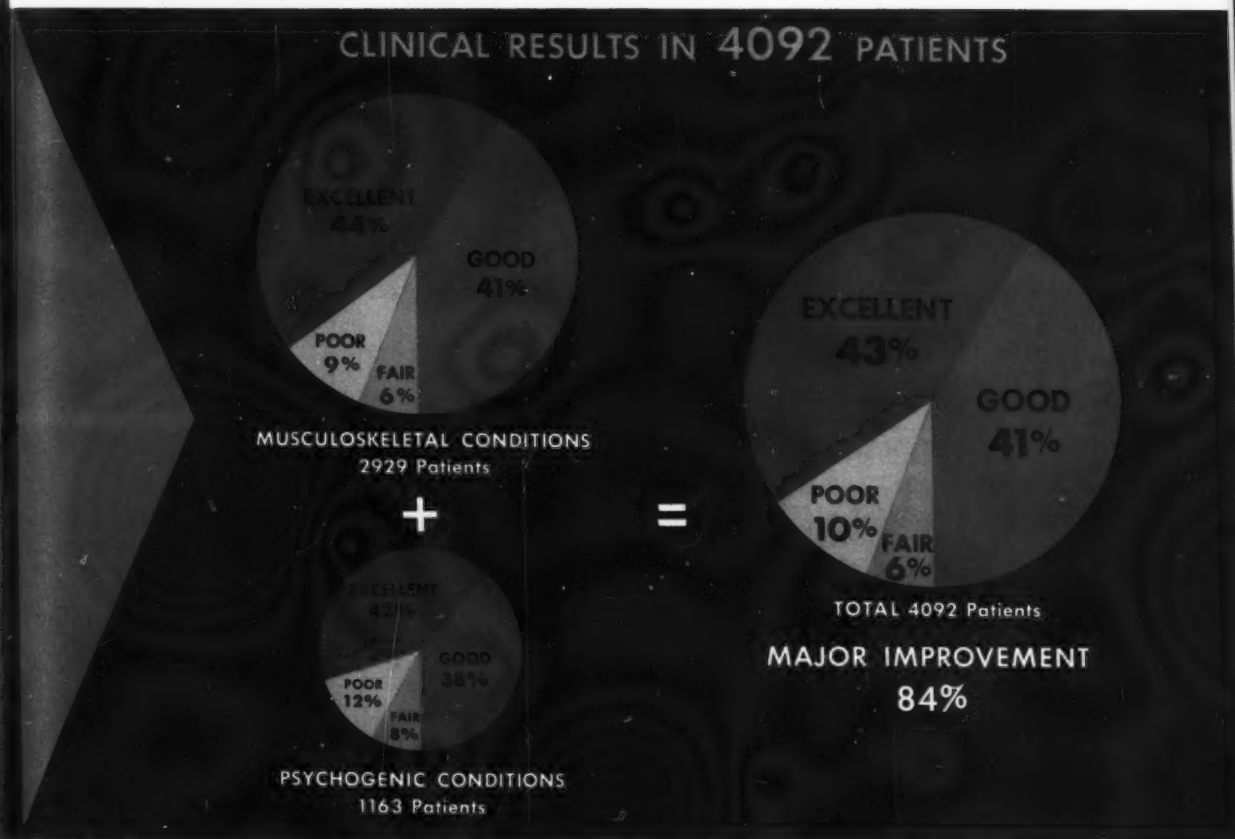
No known contraindications. Blood pressure, pulse rate, respiration and digestive processes unaffected by therapeutic dosage. No effects on hematopoietic system or liver and kidney function.

Low toxicity. In animals, even less toxic than aspirin.

No gastric irritation. Can be taken before meals.

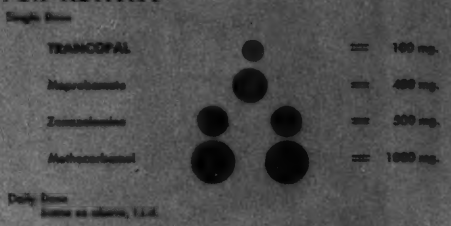
No clouding of consciousness, no euphoria or depression.

No perceptible soporific effect, even in high dosage.



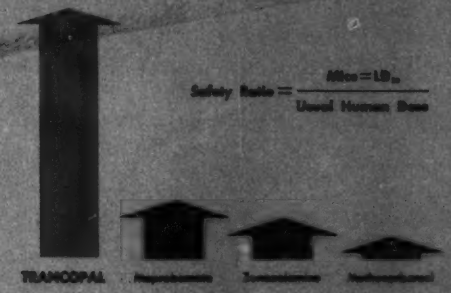
Compare Trancopal with 3 widely used central relaxants

FOR ACTIVITY



Considering the usual human dose, Trancopal, the first true "tranquilaxant," is four to ten times as potent per milligram.

FOR SAFETY



Comparative pharmacologic tests showed that Trancopal is up to thirteen times as safe, or up to thirteen times less toxic. The measure of safety was the LD_{50} in mice/usual human dose.

FOR CLINICAL EFFECTIVENESS

A clinical comparison in low back pain, torticollis, bursitis and anxiety states showed that Trancopal is up to four times as effective. Each of 40 patients received all four drugs in random rotation for several days. While each of the four drugs gave some relief, only the one providing the most effective relief was recorded.

INDICATIONS

Musculoskeletal
- Low back pain (lumbago)
- Neck pain (torticollis)
- Bursitis
- Rheumatoid arthritis
- Osteoarthritis
- Disc syndrome
- Fibrositis
- Joint disorders (ankle sprain, tennis elbow, etc.)
- Myositis
- Postoperative myalgias

Psychogenic
- Anxiety and tension states
- Dysmenorrhea
- Premenstrual tension
- Asthma
- Emphysema
- Angina

Neurologic
- Muscle spasm in paralysis agitans, multiple sclerosis, hemiplegia, poliomyelitis

TRANCOPAL thoroughly evaluated clinically

"In the treatment of conditions associated with skeletal muscle spasm there was a high percentage of satisfactory results (excellent, good or fair) in 310 patients (94%) out of 331 treated. . . . In 120 patients with simple anxiety or tension states results were satisfactory in 114 (95%). Dosage of chlormethazanone in all cases was 100 mg. t.i.d. As well as relieving the anxiety or tension state, chlormethazanone also allowed these patients to resume their usual occupations." (Lichtman)

Trancopal

the first true "TRANQUILAXANT"

Dosage: One Caplet (100 mg.) orally three or four times daily. Relief of symptoms occurs in fifteen to thirty minutes and lasts from four to six hours.

Supplied: Trancopal Caplets® (scored) 100 mg., bottles of 100.

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• Baker, A. B.: Modern Med. 26:140, April 15, 1958. • Cohen, A. I.: In preparation. • Cooperative Study, Department of Medical Research, Winthrop Laboratories. • Gesler, R. M. and Goulston, F.: Toxicol. & Appl. Pharmacol. To be published. • Gesler, R. M., and Surrey, A. R.: J. Pharmacol. & Exper. Therap. 122:24A, Jan., 1958. • Gesler, R. M., and Surrey, A. R.: J. Pharmacol. & Exper. Therap. 122:617, April, 1958. • Lichtman, A. L.: Kentucky Acad. Gen. Pract. J. 4:39, Oct., 1958. • Surrey, A. R.; Webb, W. G., and Gesler, R. M.: J. Am. Chem. Soc. 80:3469, July 5, 1958.

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the children's favorite . . .
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Vitamin C	75 mg.
Vitamin E	5 Units†
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Vitamin B-2	2.5 mg.
Vitamin B-6	1 mg.
Vitamin B-12 Activity	3 mcg.
Panthenol	5 mg.
Nicotinamide	20 mg.
Folic Acid	0.1 mg.
Biotin	30 mcg.
Rutin	12 mg.
Calcium Carbonate	125 mg.
Boron	0.1 mg.
Cobalt	0.1 mg.
Fluorine	0.1 mg.
Iodine	0.2 mg.
Magnesium	3.0 mg.
Manganese	1.0 mg.
Molybdenum	1.0 mg.
Potassium	2.5 mg.

*U.S.P. UNITS †INT. UNITS

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Supplied: Boxes of 30—one month's supply
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Miltrate is recommended *for prevention* of angina attacks, *not for relief* of acute attacks.

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Each tablet contains: 200 mg. Miltown + 10 mg. pentaerythritol tetranitrate.
Usual dosage: 1 or 2 tablets q.i.d. *before meals* and at bedtime.
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1. *Friedlander, H. S.: The role of ataraxics in cardiology. Am. J. Card.* **1**:*395, March 1958.*
2. *Shapiro, S.: Observations on the use of meprobamate in cardiovascular disorders. Angiology* **8**:*504, Dec. 1957.*

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references:

1 Grieble, H.G., and Jackson, G.G.: Prolonged Treatment of Urinary-Tract Infections with Sulfamethoxypyridazine. *New England J. Med.* 258:1-7, 1958

2. Editorial: *New England J. Med.* 258:48-49, 1958.

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REFERENCES: (1) Wise, R. I.: *J.A.M.A.* 166:1178, 1958. (2) Brown, J. W.: *J.A.M.A.* 166:1185, 1958. (3) Caswell, H. T., *et al.*: *Surg., Gynec. & Obst.* 106:1, 1958. (4) Godfrey, M. E., & Smith, I. M.: *J.A.M.A.* 166:1197, 1958. (5) Waisbren, B. A.: *Wisconsin M. J.* 57:89, 1958. (6) Royer, A., in Welch, H., & Marti-Ibañez, F.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia, Inc., 1958, p. 783. (7) Markham, N. P., & Shott, H. C. W.: *New Zealand M. J.* 57:55, 1958. (8) Blair, J. E., & Carr, M.: *J.A.M.A.* 166:1192, 1958. (9) Horan, J. M.: *Pediatrics* 19:36, 1957. (10) Rawls, C. H.: *Am. Surgeon* 23:1030, 1957. (11) Sarason, E. L., & Bauman, S.: *Surg., Gynec. & Obst.* 105:224, 1957. (12) James, U.: *Brit. J. Clin. Pract.* 11:801, 1957. (13) Turnbull, R. B., Jr.: *J.A.M.A.* 164:756, 1957. (14) Ross, S.; Puig, J. R., & Zaremba, E. A., in Welch, H., & Marti-Ibañez, F.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia, Inc., 1958, p. 803. (15) Leachman, R., & Yow, E. M., in Conn, H. F.: Current Therapy 1958, W. B. Saunders Company, Philadelphia, 1958, p. 51.

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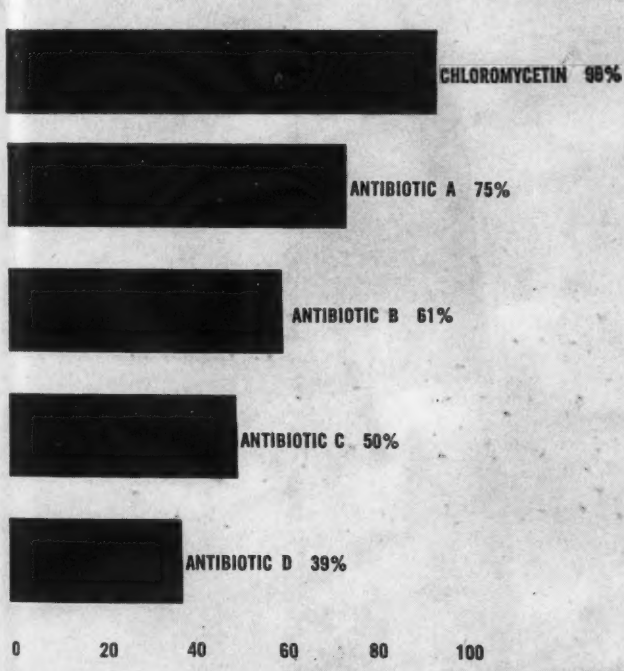
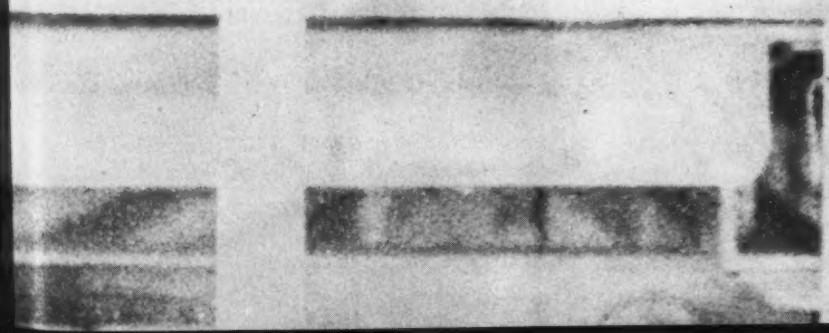
IN VITRO SENSITIVITY OF PATHOGENIC STAPHYLOCOCCI TO CHLOROMYCETIN AND TO FOUR OTHER MAJOR ANTIBIOTICS*



*Adapted from Godfrey & Smith.[4] Staphylococci studied were strains isolated from 28 patients in a general hospital.



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